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Pediatric PHARMACOTHERAPY *Self Assessment*

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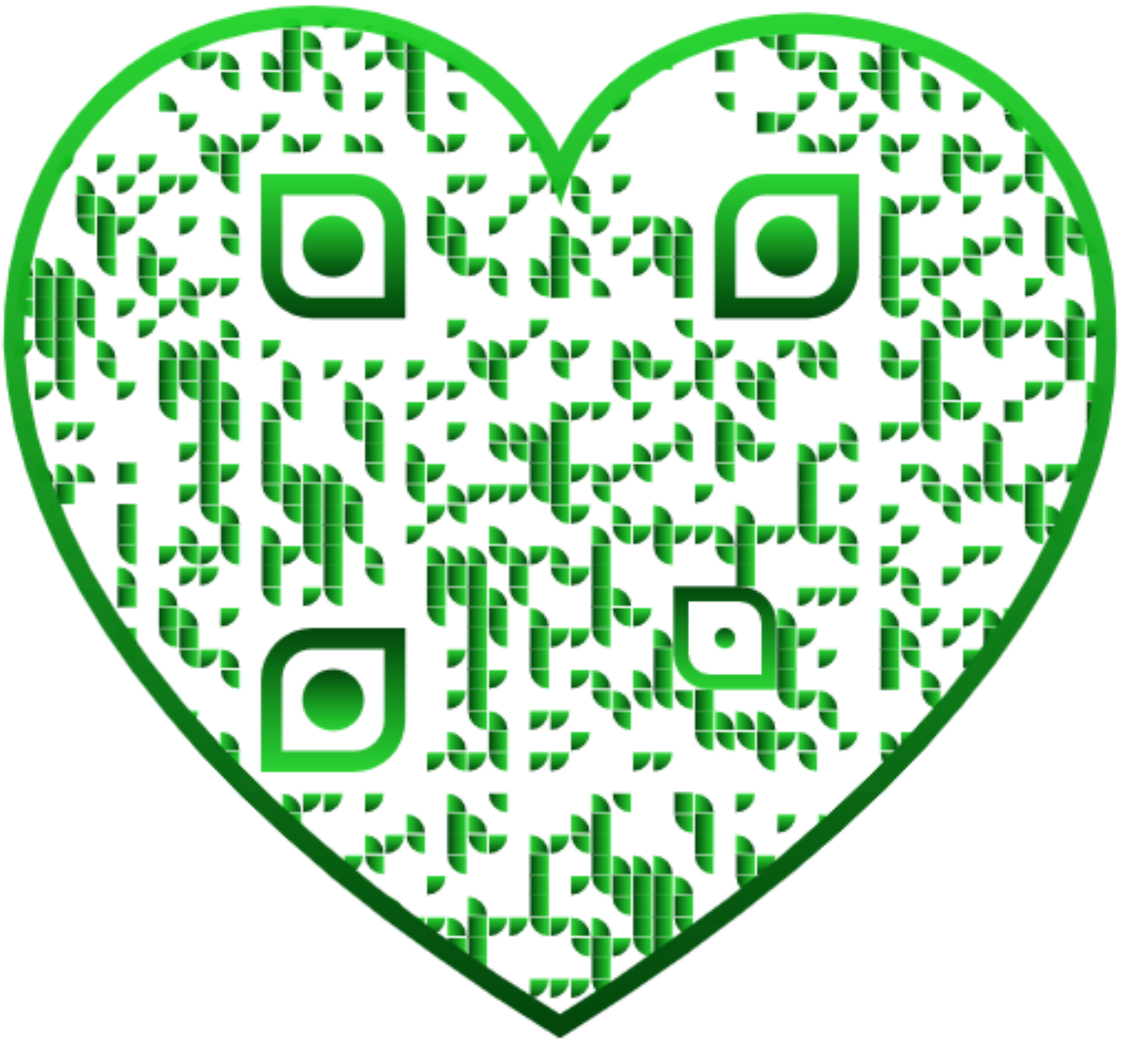
Account: s8879438



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Any correspondence regarding this publication should be sent to the publisher, American Society of Health-System Pharmacists, 7272 Wisconsin Avenue, Bethesda, MD 20814, attention: Special Publishing.

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Director, Special Publishing: Jack Bruggeman
Acquisitions Editor: Robin Coleman
Editorial Project Manager: Ruth Bloom
Project Editor: Kristin Eckles
Cover & Page Design: David Wade

Library of Congress Cataloging-in-Publication Data

Pediatric pharmacotherapy self assessment / [edited by] Sandra Benavides, Hanna Phan, Milap C. Nahata.
p. ; cm.
Includes bibliographical references and index.
ISBN 978-1-58528-424-5
I. Benavides, Sandra, editor. II. Phan, Hanna, 1979- editor. III. Nahata, Milap C., editor. IV. American Society of Health-System Pharmacists, publisher.
[DNLM: 1. Child. 2. Drug Therapy. 3. Infant. WS 366 / WS 366]
RJ560
615.1083--dc23
2014015871

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ISBN: 978-1-58528-424-5

10 9 8 7 6 5 4 3 2 1

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DEDICATION

In loving memory of Tom Bergen, whose life was made possible by treatment for a pediatric condition. He used his life to inspire us all to believe that anything is possible.

Sandra Benavides

To my family and friends, for their continued support and unconditional love—thank you for reminding me of what is important in life. To Milap, Varsha, Bob, Marie, and Sandra—words cannot fully express my gratitude for your friendship, guidance, and inspiration. To my colleagues near and far—it is truly an honor to serve with you, as advocates for our patients and families. To my students and residents—thank you for the privilege to share my passion for pediatrics with you and your ongoing thirst for knowledge.

Hanna Phan

To all those who continue to help me grow—my family, mentors, fellows, students, collaborators, patients, colleagues, and friends.

Milap C. Nahata

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CONTENTS

CONTRIBUTORS ..... **IX**

PREFACE ..... **XI**

ACKNOWLEDGMENTS ..... **XII**

SECTION 1. INTRODUCTION **1**

1.1 Pediatric Patient Care Management 1
Jaime W. Riskin and Sandra Benavides

PART I. CASES

SECTION 2. GENERAL TOPICS **17**

2.1 Anaphylaxis—Level 1 17
Nicole M. Even and Hanna Phan

2.2 Nutrition—Level 2 20
Sandra Benavides

2.3 Toxicology—Level 3 23
Mary A. Babico and Hanna Phan

SECTION 3. CARDIOVASCULAR **27**

3.1 Patent Ductus Arteriosus—Level 1 27
Brady S. Moffett

3.2 Hypertension—Level 2 29
Brady S. Moffett

3.3 Congenital Heart Defects—Level 3 31
Brady S. Moffett

SECTION 4. PULMONARY **33**

4.1 Asthma—Level 1 33
Manar O. Lashkar and Hanna Phan

4.2 Cystic Fibrosis—Level 2 36
Hanna Phan

4.3 Respiratory Distress Syndrome and Bronchopulmonary Dysplasia—Level 3 40
Hanna Phan

SECTION 5. GASTROINTESTINAL **43**

5.1 Constipation—Level 1 43
Jeremy S. Stultz and Milap C. Nahata

5.2 Crohn Disease—Level 2 46
Jeremy S. Stultz and Milap C. Nahata

continued on next page

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CONTENTS

SECTION 6. RENAL 49

6.1 Nephrotic Syndrome—Level 2 49
Sandra Benavides

6.2 Acute Kidney Injury—Level 3 52
Jeremy S. Stultz and Milap C. Nahata

SECTION 7. ENDOCRINE 57

7.1 Type 2 Diabetes Mellitus—Level 1 57
Sandra Benavides

7.2 Hypothyroidism—Level 2 60
Shirin Madzhidova and Sandra Benavides

7.3 Diabetic Ketoacidosis—Level 3 66
Julianna Crain and Sandra Benavides

SECTION 8. NEUROLOGY & PSYCHIATRY 69

8.1 Attention Deficit Hyperactivity Disorder—Level 1 69
Jennifer E. Thomas and Joshua Caballero

8.2 Autism Spectrum Disorder—Level 2 72
Jose Valdes and Jose A. Rey

8.3 Depression—Level 2 75
Kristen Lamberjack and Christine Prusa

8.4 Cerebral Palsy—Level 3 78
Mary Worthington

8.5 Seizure Disorders—Level 3 81
Jose Valdes and Jose A. Rey

SECTION 9. INFECTIOUS DISEASES 85

9.1 Acute Otitis Media—Level 1 85
Dominic Chan and Jennifer Le

9.2 Skin and Skin Structure Infections—Level 1 87
Jennifer Le

9.3 Parasitic Infections—Level 2 90
Heather L. Girand

9.4 Pneumonia—Level 2 92
Dominic Chan and Jennifer Le

9.5 Kawasaki Disease—Level 3 95
Jennifer Le

9.6 Human Immunodeficiency Virus Infection—Level 3 99
Heather L. Girand

continued on next page

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Account: s8879438

CONTENTS

9.7 Meningitis—Level 3 102
Heather L. Girand

SECTION 10. HEMATOLOGY/ONCOLOGY 105

10.1 Anemia—Level 1 105
Tracy M. Hagemann
10.2 Sickle Cell Disease—Level 2 108
Tracy M. Hagemann
10.3 Anticoagulation—Level 3 111
Brady S. Moffett

PART II. QUESTIONS & ANSWERS

2.1 Anaphylaxis—Level 1 117
2.2 Nutrition—Level 2 120
2.3 Toxicology—Level 3 126

3.1 Patent Ductus Arteriosus—Level 1 131
3.2 Hypertension—Level 2 133
3.3 Congenital Heart Defects—Level 3 136

4.1 Asthma—Level 1 139
4.2 Cystic Fibrosis—Level 2 143
4.3 Respiratory Distress Syndrome and Bronchopulmonary Dysplasia—Level 3 148

5.1 Constipation—Level 1 153
5.2 Crohn Disease—Level 2 157

6.1 Nephrotic Syndrome—Level 2 163
6.2 Acute Kidney Injury—Level 3 167

7.1 Type 2 Diabetes Mellitus—Level 1 173
7.2 Hypothyroidism—Level 2 176
7.3 Diabetic Ketoacidosis—Level 3 183

8.1 Attention Deficit Hyperactivity Disorder—Level 1 187
8.2 Autism Spectrum Disorder—Level 2 190
8.3 Depression—Level 2 195
8.4 Cerebral Palsy—Level 3 199
8.5 Seizure Disorders—Level 3 203

9.1 Acute Otitis Media—Level 1 209
9.2 Skin and Skin Structure Infections—Level 1 213
9.3 Parasitic Infections—Level 2 216
9.4 Pneumonia—Level 2 220

continued on next page

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Account: s8879438

CONTENTS

9.5 Kawasaki Disease—Level 3 223
9.6 Human Immunodeficiency Virus Infection—Level 3 227
9.7 Meningitis—Level 3 233

10.1 Anemia—Level 1 239
10.2 Sickle Cell Disease—Level 2 242
10.3 Anticoagulation—Level 3 247

APPENDIXES

Appendix 1: Data Collection Sheet 254
Appendix 2: Common Medical Terminology and Abbreviations in Pediatric Practice 257
Appendix 3: "Do Not Use" Abbreviations List 260
Appendix 4: SOAPE Outline—Multiple Days or Visits 261
Appendix 5: SOAPE Outline—Single Day or Visit 265

INDEX 267

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PREFACE

Pharmacists providing pediatric patient care require unique knowledge and skills. Pharmacists must evaluate patient-specific data such as clinical presentation, medical history, and laboratory or diagnostic tests to formulate an appropriate therapeutic plan. Of special importance in pediatric patients are age, physiological development, body-weight specific dose requirements, dosage form availability, medication administration, and caregiver education in the clinical decision-making process. *Pediatric Pharmacotherapy Self Assessment* is designed to provide patient-specific, case-based learning opportunities for students as well as new and advanced practitioners. The casebook will afford opportunities to apply pharmacotherapy knowledge in the care of neonates, infants, children, and adolescents. An assortment of patient cases, ranging from acute to chronic pediatric conditions, with varying complexities have been included with corresponding self-assessment questions and answers.

Each case was designed to include patient-specific data for evaluation to formulate specific recommendations for all aspects of care including assessing current treatment, identifying an alternative treatment when necessary, developing goals and clinical outcomes, devising monitoring parameters, and selecting the most pertinent and appropriate patient/caregiver education. The intent of each case is to develop and hone skills in identifying subjective and objective data to support a specific diagnosis and critically evaluate current pharmacotherapy, including identification of medication-related problems to self assess knowledge or skills. The self-assessment cases may be useful prior to or during didactic or pediatric-specific elective courses or an introductory/advanced pharmacy practice experience (IPPE/APPE) involving pediatric patients. This casebook's varying levels of difficulty allow further self assessment after attainment of further knowledge and skills.

The first chapter provides a detailed discussion of the clinical assessment of pediatric patients highlighting differences in this population. The chapter includes examples of methods toward patient-specific data collection, evaluation, and development of a therapeutic plan. Beginning with Section II, clinicians and educators have contributed a variety of cases to simulate real-world scenarios often encountered in practice. Cases are designed as beginner (level 1), intermediate (level 2), or advanced (level 3), which allows the casebook to be a resource for students, residents, and practitioners. Commonly used medical terminology and abbreviations are provided throughout the book to familiarize the reader about the practice of pediatrics. Because multiple approaches are possible, experienced practitioners have provided treatment approaches based on primary literature, guidelines, and clinical experience. A bibliography is included with each case to direct the reader to additional resources for each topic. The casebook has incorporated many unique characteristics to consider when caring for pediatric patients. Additionally, most cases incorporate a medication-related problem for the reader to identify and formulate a solution. The intent of the editors and contributors is to provide opportunities for self assessment through practice and knowledge application, thereby contributing to the development of practitioners to offer the best possible care for children.

Sandra Benavides

Hanna Phan

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ACKNOWLEDGMENTS

The editors cannot express enough gratitude to the contributors involved in *Pediatric Pharmacotherapy Self Assessment*. They developed practical cases from professional experience with pediatric-specific considerations important in the care of neonates, infants, children, and adolescents. The editors appreciate the time they took to develop the cases, self-assessment questions and answers. We know these will be a tremendous asset for students and practitioners in developing skills necessary to care for pediatric patients.

The editors are indebted to Manar O. Lashkar, PharmD, and Titilola "Lola" M. Afolabi, PharmD, at The Ohio State University for their thoughtful and careful review on every case. At times, they found that what the editors thought was clearly written did not read easily and clearly. Your time, efforts, and input were invaluable. We would also like to thank Elizabeth Lozada, PharmD, for her suggestions to many cases during her advanced pharmacy practice experience (APPE) at Nova Southeastern University.

We would like to express our appreciation to the staff at the American Society of Health-System Pharmacists (ASHP) for the opportunity to provide a self-assessment tool to students and practitioners. Thanks to Robin Coleman, Ruth Bloom, Kristin Eckles, and David Wade for the input, constructive comments, editing, design of the casebook, patience, and support throughout the publication process.

Most importantly, we would like to acknowledge the contributions of all practitioners in pediatrics. Those who came before us have forged a path for the specialty recognition of pediatric pharmacotherapy. The current practitioners continue to expand the areas of practice and impact health outcomes in children. Those to come will carry our banner to continue the work and progress that has been accomplished to date. We hope this casebook contributes to the development of your knowledge and skills in pediatrics for the evolution of pediatric pharmacotherapy.

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1.1 Pediatric Patient Care Management

Jaime W. Riskin and Sandra Benavides

INTRODUCTION

Patient care management is the process of identifying, resolving, and preventing health-related problems and needs. Although the focus may revolve around specific disease states, the process should remain patient-centered. With pediatric patients, the process often is family-centered as the patient relies on a caregiver to be involved in some or all aspects of healthcare. All health-related problems and needs must be addressed with definitive outcomes. As the role of the pharmacist continues to expand in direct patient care, accountability is more important than ever and clinical documentation is crucial.[1–4]

Developing a technique and critical analytical skills for the assessment of patients is an important tool for all practitioners. In pediatric practice, the approach may differ from that of the adult patient. Differences and barriers may exist in obtaining some information from a pediatric patient. For example, in infants (1 month to 1 year of age) and toddlers (up to 3 years of age), the practitioner is reliant on a caregiver to identify and communicate specific signs and symptoms the infant or toddler cannot communicate, such as pain. Often, information not obtained in adult counterparts is important in pediatric patients to fully assess the patient, such as the birth history. In this introductory chapter, pediatric-specific differences are highlighted and strategies are presented to assist the reader to be successful in assessing this population.

As pharmacists, the focus of our assessments is often related to drug therapy, disease state management, and preventive strategies (e.g., immunizations). We are trained to identify if a particular medication is the most appropriate for a patient to achieve a desired outcome or endpoint while minimizing adverse reactions or toxicity. The pharmacist also identifies medication-related problems (MRPs) such as drug interactions, duplicate therapy, or contraindicated drugs. In the process of gathering and analyzing patient-specific data, the pharmacist remains focused on the optimization of clinical outcomes. The cases in this book will assist the reader in identifying and providing solutions to health-related problems and needs in pediatric patients.

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ASSESSMENT OF MEDICATION EFFICACY AND SAFETY

A pharmacist specializing in pediatrics is uniquely trained to develop, assess, and monitor medication regimens for optimal efficacy and safety. The knowledge of differences in absorption, distribution, metabolism, elimination, and pharmacodynamic effects of medications in a variety of disease states specific to the pediatric population provides a valuable contribution to the healthcare team. The American Academy of Pediatrics has long supported the inclusion of a pharmacist trained to care for pediatric patients in interprofessional teams in pediatric and neonatal intensive care and hematology/oncology settings.[5–7] The role of the pharmacist is further expanding into various ambulatory and community venues. Pharmacists specialize in pediatric pulmonology, nephrology, endocrinology, and other subspecialties.[8] Regardless of the healthcare setting, the pharmacist's role is to optimize pharmacotherapy, minimize adverse drug reactions, or offer appropriate and effective preventive strategies to improve the health of the patient.

Pharmacists begin assessing a patient by performing a global assessment to identify general health-related needs. For example, during an encounter, a pharmacist may ask the child or caregiver about immunization status. During the interview, the pharmacist may note a specific recommended immunization was missed, such as a tetanus booster. After the global assessment, the pharmacist will identify and assess individual health-related needs, including specific disease states. In this instance, a pharmacist may have an encounter with a patient diagnosed with type 1 diabetes mellitus and recommend or administer (in some states) the influenza vaccine because of the patient's high risk for pneumonia secondary to the flu.

In reviewing each health-related need or problem, the pharmacist focuses on appropriate use, efficacy, and adverse reactions of medications. In doing so, the pharmacist is able to characterize not only health-related problems or needs, but also to identify and propose solutions to MRPs. An MRP can include inappropriate use of a drug, development of an adverse reaction, or taking an agent for an unclear indication. An MRP is made up of three components: the actual or potential event, the drug suspected to be the cause of the problem, and the relationship between the drug and the event. The relationship is the proposed cause and effect, which can help determine the proposed resolution of the problem (e.g., discontinue or initiate a medication). **Table 1-1** includes examples of MRPs and potential causes.

A systematic approach to the identification and resolution of health-related problems or needs in order to develop and execute a patient care plan has been proposed.[4] The first step is to identify the suspected problem or need. Once identified, establishment of patient-specific treatment goals and outcomes are necessary in order to develop an appropriate care plan. Potential treatments are identified and selected to design a patient care plan. Monitoring parameters to evaluate resolution of the problem is addressed with the plan, including presenting signs and symptoms, laboratory data, and subsequent follow-up visits with the patient, when appropriate. Depending on the clinical setting, the plan is either communicated with the medical team, another healthcare provider, or directly with the patient. It is imperative to educate the patient or caregiver on changes to therapy. The final step is documentation of the plan. Without appropriate documentation, there is no record of the event occurring, which is vital for other providers caring for the patient. Additionally, the medical documentation may be necessary if legal proceedings were to occur.

GATHERING MEDICAL INFORMATION

The first step to assessing the pediatric patient is gathering pertinent medical information. Depending on the setting, this process occurs by reviewing medical records, interviewing the patient or caregiver(s), and performing general patient assessment techniques. Gathering the appropriate and necessary information will assist the practitioner in assessing medication effectiveness and safety, providing preventive health recommendations, and aid in the management of medical conditions.

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TABLE 1-1: MEDICATION-RELATED PROBLEMS

Medication-Related Problem Category	Example
Medication or therapy needed	Patient with chronic ear infections due to allergies who needs a daily antihistamine
	A 2-month-old infant who has not received the first round of the hepatitis B vaccine
Inappropriate medication	Mother administers diphenhydramine to her child to sleep through the night
	Patient receiving both pantoprazole and ranitidine for acid reflux
	Adolescent taking father's alprazolam for recreational use
Wrong dose	Obese child given a weight-based dose of digoxin that exceeds the adult dose
	Tacrolimus administered once daily rather than twice daily after a liver transplant resulting in rejection
Alternative product or formulation needed	Toddler with stomach virus who is unable to tolerate oral intake would benefit from a rectal acetaminophen suppository, rather than the liquid
	Mother who often forgets doses of her child's ADHD medication, which is dosed tid, would benefit from a once-daily extended-release formulation
Drug interaction (drug–drug, drug–food, drug–disease)	Ceftriaxone prescribed to a premature neonate receiving calcium in his parenteral nutrition
	Infant prescribed erythromycin for pertussis who is already taking sildenafil for pulmonary hypertension
Safety issues	9-year-old child treated with an antidepressant who develops suicidal ideations
	Hives after starting sulfamethoxazole–trimethoprim for a community-acquired MRSA skin infection
	Fungal diaper rash as a result of high-dose amoxicillin for an ear infection

ADHD = attention deficit hyperactivity disorder; MRSA = methicillin-resistant *Staphylococcus aureus.*

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REVIEWING MEDICAL RECORDS

Different clinical settings will provide varying amounts of the patient's medical, personal, and social histories. In the most extreme case, a pediatric intensive care unit (PICU) medical record will typically have immense amounts of data ranging from a detailed History and Physical (H&P) to hourly vital signs. Reviewing such records can be cumbersome and—to a novice practitioner—overwhelming. It is important to take time to review the medical record (either paper or electronic) to familiarize oneself with the layout and the data available.

Initially, early practitioners often gather voluminous amounts of information until they fine tune the skills to identify the pertinent and salient patient-specific information. Reviewing the medical record in chronological order will tell the medical story of the patient and will assist in identifying health-related problems (both ongoing and resolved). Documentation of administration of medications via the Medication Administration Record (MAR) is typically accurate. Although many data may be available for the patient's current illness, information regarding chronic conditions and treatments may not always be readily available. Appendix 1: Data Collection Sheet provides a worksheet for gathering medical data in an organized manner.

Practitioners in the ambulatory care setting may be more limited in the information they can access. The medical records may include progress notes from the primary or specialty providers. Vital signs and laboratory data may not be recent. Often, medical reports

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and laboratory and diagnostic test results have to be obtained from various sources in order to provide a complete assessment of the patient. Medications listed in the medical record may be incomplete or inaccurate. Furthermore, the ambulatory medical record does not provide information on whether the medications are administered, and the assessment of adherence is dependent on interviewing the patient.

Perhaps the practitioner with the most limited access to medical records is the community pharmacist. In this setting, often the only data available to the patient are basic patient demographics and the medications dispensed. Similar to the ambulatory setting, the medical record in the community can span a number of years, providing the practitioner a year-by-year account of the individual's medical problems and past treatments. Some pharmacists in this setting do collect more detailed information, such as family and personal history, alcohol and tobacco use, and ongoing medical conditions at each encounter; however, this is not considered commonplace. As such, the community practitioner must rely on other methods of obtaining a complete medical history, such as interviewing the patient or caregiver, point-of-care testing, or physical assessment. The medication records in this setting may not be complete, as patients may have obtained medications from other sources. Although the community pharmacist can identify nonadherence through missed refills, adherence must still be assessed through the patient or caregiver interview.

Regardless of the setting, caution must be taken when placing too much reliance on the medical record. The paper or electronic medical records provide insight into the probable medical conditions and needs of the patient but may not be complete and at times may be inaccurate. One study in a pediatric epilepsy clinic revealed that nearly 50% of all medications listed in the medical record were incorrect or did not reflect how the patients were taking the prescribed medications.[9] This underscores the importance of obtaining a detailed medication history.

CONFIRMING A MEDICATION HISTORY

A complete and accurate medication regimen is vital to ensure a continuum of care across all healthcare settings thereby optimizing the patient's outcomes.[10,11] Documenting current medication regimens is necessary to assess appropriateness of therapy (including treatment goals and outcomes), continue or alter therapy, monitor the patient for efficacy and toxicity, and identify adverse drug reactions. A medication history includes chronic prescription medications, over-the-counter medications, herbal products, nutritional supplements, and recent short-course treatments such as antibiotics, corticosteroids, and pain medications. Medications taken on an "as needed" (prn) basis should include documentation of the reason for taking the medication and the frequency of administration. When possible, the start date of the medication should be included. For chronic or serious conditions, such as epilepsy, malignancies, or human immunodeficiency virus (HIV), it is useful to document all past medication regimens, the dates of initiation and discontinuation of a drug, and reasons for discontinuation (e.g., adverse drug reaction, incomplete efficacy). Lastly, a review and documentation of all drug, food, and environmental allergies with the specific reaction should be conducted at each encounter.

RECONCILING MEDICATIONS, INCLUDING TRANSITION OF CARE

Medication reconciliation (often referred to as *med rec*) is the process of obtaining a complete medication history in collaboration with a patient or caregiver to provide an accurate medication list to healthcare providers at any transition of care.[12] Although all healthcare providers may be responsible for medication reconciliation, the pharmacist may be the best equipped to obtain a complete list and immediately begin identifying problems due to the focus of his or her education. In order to perform a proper reconciliation, it is vital that an accurate medication history be obtained at each patient encounter. This process allows the

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healthcare team to improve the medication use process for patients and identify and prevent more MRPs. In pediatric patients, limited data have been published regarding medication reconciliation and all is focused on the acute care setting (admissions, discharge, and transitions between levels of care).[13] Rates of discrepancies in the medication histories to what was actually prescribed ranged from 22% to 72% at admission and 15% at discharge.[13] These studies highlight the need to establish strategies to conduct medication reconciliation in all healthcare settings.

DOCUMENTATION

Demonstration of pharmacy contribution to patient care is accomplished via accurate and complete clinical documentation. Patient outcomes, cost-effective initiatives, reimbursement, and legal or regulatory issues are addressed via documentation. According to the American Society of Health-System Pharmacists (ASHP), any professional action by a pharmacist to ensure safe and effective medication use and potentially affect outcomes should be documented in the patient's medical record.[2,3] As such, in some institutions and settings, pharmacists document consultations on medication management, renal dose adjustments, changes to route of administration (i.e., intravenous to oral therapy), patient counseling and education, drug therapy monitoring, medication history and allergies, and clarification of physician orders in the medical records. Clinical interventions should be recorded in a permanent manner to be available to all members of the healthcare team to ensure continuity of care. Healthcare systems should establish policies and procedures that delineate the authority of a pharmacist to document in the patient's medical record and determine a consistent location for such documentation.

Clinical documentation also ensures standardization of care among various healthcare professionals, particularly regarding patient clinical outcomes. Communication between professionals, whether it is intra- or interprofessional, is accomplished through clinical documentation.

Electronic health records (EHRs) are becoming more commonplace and expected to become more so in both inpatient and outpatient settings. It is important to understand the benefits and limitations of EHRs. The positive attributes include legibility as well as ease and availability of use. Often, EHRs are Internet-based and can be accessed remotely. EHRs typically use protocols and drop-down menus, allowing for standardized, evidence-based medicine. Lastly, EHRs have the ability to provide warnings (often referred to as *flags*) and clinical decision support.

However, as with any new technology, opportunities for improvement still exist with many EHRs. As healthcare systems implement new systems, there is an increased need for training the users of the system due to the lack of familiarity by most. Some institutions may use multiple systems during the transition and require consolidation into one system. In some of the available EHRs, medication orders are often displayed separately from others, such as laboratory and nursing orders; therefore, it can be difficult to follow the thought process of the prescriber. EHRs have been criticized as providing "cookie cutter" medicine, as they may create a potential to lose some of the patient-specific care plan attributes. There is a potential to select the incorrect option from a drop-down menu and drug information varies with different venues, often time neglecting pediatric-specific monographs. Finally a phenomenon called *alert fatigue*, where the user of the EHR begins to bypass the alerts without reading or acting on them, creates a potential for medication errors, despite the provision of flags and clinical decision support. Accurate clinical documentation, despite some limitations of EHRs, is extremely valuable.[14,15]

Clinical documentation of a patient care plan is accomplished by writing a note, which must be clear, concise, consistent, and complete.[2,3] The note should be user-friendly, in that it should be easy for any healthcare provider to gather information quickly and accurately by knowing where to find information within the document. Notes should be structured and organized, legible, and use common vocabulary that is understood across disciplines (see Appendix 2: Common

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Medical Terminology and Abbreviations in Pediatric Practice for a list of commonly used abbreviations and Appendix 3: "Do Not Use" Abbreviation List for those that are unapproved in hospital settings).[16] It must also be patient-focused.

There are various systematic documentation styles. The most universal approach is the SOAPE format (**s**ubjective, **o**bjective, **a**ssessment, **p**lan, **e**ducation). Others include TITRS (**t**itle, **i**ntroduction, **t**ext, **r**ecommendation, **s**ignature), which is an assessment approach, and FARM (**f**indings, **a**ssessment, **r**ecommendations/resolutions, **m**anagement), which focuses on monitoring. The SOAPE format is an interventionist approach, in that it is all-encompassing and provides the evidentiary information, an assessment, a plan, and follow-up with monitoring.[3] This casebook will utilize the SOAPE style throughout. Appendix 4: SOAPE Outline—Multiple Days or Visits and Appendix 5: SOAPE Outline—Single Day or Visit provide a skeleton for the SOAPE note for a patient visit or for longitudinal patient visits.

SOAPE NOTES

SOAPE notes were initially developed in the 1970s as a physician-to-physician communication tool. The format has since been adopted and customized by other healthcare professionals. A pharmacy-specific modification is the inclusion of *recommendations* versus *plan* depending on the autonomy of the pharmacist. For those working within a collaborative drug therapy management agreement, the plan may be implemented; whereas, those who do not should include a recommendation to a prescriber to make the change.

Demographic Patient Data

Demographic information, such as the medical record number, date of birth, age, gender, ethnicity, primary language spoken, caregiver's name, insurance status, and address and/or room number, should be noted and included prior to the subjective section in the note.

Subjective Data

Subjective data are findings reported by the patient or caregiver. Examples of subjective data include presenting symptoms, past medical history, family and social history, medication, food and environmental allergies, and past experience with medications. Subjective data require further verification through objective data such as medical records or a physical exam. For example, the medication history and allergies may be listed in the subjective data; however, once verified through pharmacy dispensing records, it then becomes objective data. The information provided by the patient or caregiver assists in identifying which additional information is necessary to identify health-related issues, select appropriate medications, and develop therapeutic solutions. Subjective information also allows the healthcare professional to formulate a hypothesis of the patient's potential issues and more directly focus further examination.

Standard components of the subjective sections include the chief complaint (CC), history of present illness (HPI), past medical history (PMH), family and social histories (FHx and SHx, respectively), review of systems (ROS), immunizations, medication history, and allergies.

The CC is the reason for the patient's visit. It is often documented as a direct quote, especially if relating to a specific symptom. Examples include "my son has been complaining of a headache" or "my daughter has had a fever of 103°F." Regularly scheduled appointments, such as a follow-up visit, would be considered the CC (although it is not technically a complaint, per se). For example, "The patient is here for a refill of her antiepileptic medication." If one is to consider the SOAPE note as a story, then the CC is considered the title of this story.

The HPI describes the circumstances surrounding the CC. The HPI requires further probing and provides information regarding onset and duration of the event, exacerbating or relieving factors, concomitant or comorbid conditions that may play a role in the event, and other pertinent information to further explain the event. With regard to the story analogy, the HPI would be the background or introductory paragraph.

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The PMH is a list of all conditions, diseases, procedures, and surgeries that are pertinent to the patient's health history. Although it is called the *past* medical history, it should include current conditions as well. It is important to include the date of onset or duration of all conditions and surgeries, as indicated as similar to the medication list.

If a condition is mentioned in the HPI, it is sometimes excluded from the PMH. Some healthcare providers may still prefer to include it in the PMH, because, as mentioned earlier, the note needs to be user friendly and clear. By documenting some conditions in the HPI and others in the PMH, a practitioner would have to look in multiple places to gather all of the desired information, which could be time-consuming and confusing.

Many times, the birth history (including any complications) will be listed in the PMH in pediatric patients up to 5 years of age, as factors related to the birth history may contribute to ongoing medical issues. Developmental history for infants and young children including age of crawling, walking, and first words, is important for identifying any motor or cognitive delays. Some practitioners will also document any specific dietary considerations. In newborns and infants, this may include whether the infant is breastfed or on infant formula, including the quantity.

Immunization status is generally included in a SOAPE note for pediatric patients. It may be listed as part of the medication history or as a separate section. Most often, caregivers are asked if the immunizations are up to date (UTD) or not, including seasonal immunizations such as influenza. Ideally, a record of all immunizations should be documented in the medical record and easily accessible.

The FHx is pertinent information about the immediate family's medical history; if a death has occurred, the FHx should include the relationship, age, and cause of death of the particular family member. Certain disease states, such as leukemia, may require gathering a deeper history beyond immediate family members; therefore, it is important to consider the condition in which the information is listed for.

The SHx is any information related to the patient's social situation. In younger patients, this section typically includes family members (e.g., number and age of siblings), the living situation of the patient (e.g., extended family members in home), presence of typical allergens in the home, such as pets, smoking history of anyone in the home, whether or not the child attends daycare or school, and general performance in school. In adolescents, the use of tobacco, alcohol, or illicit drug use should also be included.

The ROS includes patient-reported symptoms, which are grouped by organ system. It should be approached in a head-to-toe manner, which is most logical. Again, this is subjective information that must be validated with additional findings, such as the physical exam or laboratory results. One should not create an assessment and plan based on the patient's reported symptoms. For example, a caregiver may report new-onset diarrhea after starting an antibiotic. Without further workup, an assessment may presume the diarrhea is due to the drug and the plan could include changing or discontinuation of the medication, when that may not actually be the case. A full evaluation of recent sick-exposure would be necessary to rule out infectious diarrhea.

Objective Data

Objective data are observations and information gathered by a healthcare provider. These findings should verify and further explain the subjective findings. The pharmacist's objective section should also record other healthcare professional's assessments and plans, such as physicians, nutritionists, and physical therapists.

Standard components of the objective sections are the physical examination (PE), which includes vital signs (VS), laboratory values, and diagnostic tests. Others may also include indwelling medical devices, such as gastrostomy tubes and venous access (e.g., central line).

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The PE is the traditional documentation of physical assessment findings. The PE can be conducted by any healthcare practitioner who is certified to do so, including appropriately trained pharmacists. Commonly used abbreviations are often included, and it is important that they are understood and interpreted properly (see Appendix 2: Common Medical Terminology and Abbreviations in Pediatric Practice). When assessing a patient for any health-related problem, it is important to address any physical findings that are relevant or significant.

The VS section is an important part of the PE but may be listed separately, immediately preceding the PE. Vital signs include blood pressure (BP), heart rate (HR; or pulse [P]), respiratory rate (RR), and temperature (T) including route obtained. Pain (sometimes referred to as the *fifth vital sign*) and oxygen saturation (SpO_2) may also be included. The documentation of pain in the pediatric patient should be made using a validated pain scale such as the Neonatal Infant Pain Scale (NIPS), Face, Legs, Activity, Cry, Consolability (FLACC) scale, Wong-Baker FACES® Pain Rating Scale, or a visual analog scale (VAS). Anthropometric assessments are often documented as part of the VS section, although in reality they are not true vital signs. Such parameters include the height, weight, body mass index (BMI), and head circumference (in infants). More importantly, the corresponding percentiles based on appropriate growth charts should be included with the values to appropriately assess nutrition and health status.

Laboratory results (referred to as *labs*) are important to obtain for the diagnosis of a condition and to monitor response to treatment. The laboratory results should include any findings pertinent to the condition and can include serum drug concentrations and microbiological culture and sensitivity reports. Labs may be ordered that appear unrelated to the condition; however, they are important in the selection of appropriate drug therapy (e.g., renal function). The results that are included in the SOAPE will depend on the nature of the patient's visit or consultation. For example, an initial consult may ask for a recommendation for a medication regimen in a newly diagnosed type 1 diabetic adolescent. For this patient, you may need to document only the most recent and current laboratory findings. However, if the patient is presenting for an assessment of his current medication regimen for the treatment of his diabetes, data from multiple days (e.g., self-monitoring blood glucose) may be included in the SOAPE to document trends necessitating change. It is important to note that laboratory values may differ based on age and gender, and pediatric references should always be used when available. For example, serum creatinine in infants is less than adults due to differences in skeletal muscle mass.

Diagnostic tests (Dx tests) include diagnostic imaging, electroencephalogram (EEG), sleep studies, and other physical tests that can help to elucidate or confirm a health problem as well as MRPs. Sometimes Dx tests will be listed as a part of the labs section but should preferentially be separated out.

Assessment

The *assessment* section of the SOAPE should be structured as a prioritized problem list. A useful way of prioritizing is to divide the complete list of identified health-related problems and needs into three subsections: (1) those that are most important and must be addressed immediately; (2) those that must be addressed during this visit; and (3) those that should be addressed but can wait until after this visit.[17] Life-threatening or acute issues, including the reason for the hospitalization or clinic visit and any newly identified issues, should be prioritized to either the first or second tier. Any abnormal findings in the PE, labs, or Dx tests must be assessed for urgency. Controlled chronic issues or unusual findings that are not pressing can be considered third-tier problems.

The assessment should be based on subjective and objective evidence. As in a court case, the judge cannot make a decision based on simple accusations but rather requires actual proof that corroborates the statements. For example, a patient taking calcium carbonate for hypoparathyroidism may state he is adherent to the medication. However, the

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serum calcium concentrations from the most recent laboratory parameters have dropped from previous results. In this scenario, one has the evidence that the patient is not taking the medication as prescribed. It is important to identify a proposed etiology for the MRP (e.g., nonadherence) and to address current therapy (or lack thereof) to determine whether or not there is need for a dose escalation, new, or additional therapy. Without identifying the etiology or a probable cause for the problem, it becomes difficult to establish a plan for correcting or preventing it. For the example above, if the healthcare provider did not probe the child on adherence to the medication, the dose may inadvertently be increased. In this situation, after further questioning, the child reveals he is no longer taking the calcium carbonate because his mother bought a new flavor that he does not like. Discrepancies between subjective and objective findings must be addressed in the assessment section. Any mismatches between medications and their use should be addressed as well. Another important point is to maintain professionalism and to avoid accusations, belittling, judgment, or complaining when writing the assessment section (e.g., the child is being difficult and picky about the flavor of the medication).

Plan and Education

The *plan* is the management strategy. Once the health-related problems and needs are identified and prioritized, a plan is developed to address each issue. The plan should be patient-specific and evidence-based. When recommending a specific medication, the drug name (preferably the generic drug name), dose, titration schedule, route, and frequency of administration, must be provided. If the dose is to be administered on a prn basis, specific criteria for use should also be provided.

The plan should include the goals or desired outcomes of therapy. The ultimate goal of pharmacotherapy is to successfully treat or prevent a condition while also minimizing adverse reactions. Specific therapeutic endpoints are established to determine if the goal is met. For example, in a patient with acute otitis media, the goal is to eradicate the infection. However, in order to objectively determine that the bacteria were eradicated, a tympanocentesis would be necessary. Such procedure is not the standard of care in the treatment of acute otitis media. Therefore, surrogate markers are used as monitoring parameters to assess if the treatment is meeting the desired outcomes of therapy. For example, reduction of fever, resolution of pain, and normalization of the child's ear examination are surrogate markers for the goal of eradicating the infection. If the therapeutic goal is not documented, it is not possible to recognize treatment success or failure. Such specific monitoring parameters, including frequency, aimed at assessing such specific goals are listed in the plan.

The plan can include any treatment strategy including pharmacologic or nonpharmacologic therapies, the need for additional diagnostic testing, follow-up time frames, and consultations. If a problem is assessed as a stable condition, then the plan would be to continue the same, monitor as indicated, and ensure the patient or caregiver is educated on the key points of the plan.

Next steps for treatment in the plan is useful for continuity of care. For example, a patient may be initiated on a new blood pressure medication that must be slowly titrated based on BP. The next step would state that if the BP has not dropped by a certain amount, then the medication should be increased. Equally important are the backup plans and they should be documented in addition to the primary plan for the case of treatment failure or unexpected situations such as drug availability or adverse drug reactions. By including another option, treatment can be conducted in a more efficient and expedient manner.

In some instances (e.g., acute care setting) the plan is often discussed with the primary physician or medical team caring for the patient. Once the plan is discussed and agreed on, it is useful to document, "Plan discussed with and agreed on by medical team," for improved communication between all healthcare providers for the patient. In the ambulatory care setting, a statement regarding communicating the plan to the primary care physician is also important.

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Education to the caregiver or patient should be a part of any plan; however, many choose to separate it out within the note. The education section of the note includes the provision of information to the patient or caregiver regarding medication and lifestyle modifications. Information such as indication, potential common and serious adverse reactions, and appropriate situations to seek additional medical care should be included. The patient or caregiver should be educated regarding signs and symptoms of success and failures. It may be helpful, especially at transitions of care, to separate the education section into "Medications That Are to Be Continued," "Medications That Will Be Changed or Discontinued," and "New Medications." Education can also apply to other members of the healthcare team. For example, if a medication requiring frequent monitoring for serious adverse reactions is initiated, the nurse should be alerted.

COMMUNICATION WITH PEDIATRIC PATIENTS

Communication with pediatric patients in any setting is vital to obtain necessary information to determine what problems may exist and to provide the patient with the appropriate information to implement a plan. Communication should be bidirectional with the ability of both the healthcare provider and the patient to obtain the information they need. For a pharmacist, the initial interaction may be geared at obtaining a complete and accurate medication history. Ideally, asking the caregiver or child to list all the medications with dose, route, and frequency is ideal; however, if the caregiver or child is unable to do so, it is appropriate for the pharmacist to verify medications from a recent medication record. In addition, the pharmacist may have to call a community pharmacy to get a record of medications for chronic conditions. After obtaining this information, the pharmacist can then ask the patient what concerns or problems surrounding medication use currently exist. At this point, the use of an open-ended question such as, "What problems, if any, are you having with your medications today?" would be more appropriate than a close-ended question such as "Are you having any problems with your medications?" Caregivers and children in particular have the tendency to want to please the interviewer and may ignore any ongoing concerns. Following the collection of the medication regimen, the communication from the pharmacist will shift from questions about medication regimens to education about a specific treatment plan.

Throughout the entire interaction with the patient and caregiver, the pharmacist will address adherence and may employ motivational interviewing techniques. *Motivational interviewing* is a mechanism for communication in which the healthcare providers influence patients to choose to follow their own customized therapeutic plan. Motivational interviewing can improve patient outcomes via adherence to therapeutic regimens (including healthy lifestyle changes). Patients must always be the core of the treatment care plan. In order to ensure adherence, patients must be willing and motivated to participate in their own healthcare. It is vital that the healthcare provider establish rapport with the patient to build a safe and comfortable environment in which the patient can open up and share concerns.[18] The five principles of motivational interviewing have often been referred to as "READS" (**r**oll with resistance, **e**xpress empathy, **a**void argumentation, **d**evelop discrepancy, and **s**upport self-efficacy).[19] **Table 1-2** has examples of each principle and how they can be applied when communicating with pediatric patients or caregivers.

The U.S. Pharmacopeial Convention (USP) has long advocated for medication information to be communicated to children.[20] Beginning at 2 years of age, it is appropriate and encouraged to include the child in a discussion regarding medications and healthcare. By beginning communication and involvement at a young age, a child may be a more active participant in his or her own healthcare. The level of interaction and depth of information obtained and provided to the child will differ based on age, experience with the healthcare system (e.g., chronic diseases), and cognitive ability. As such, healthcare providers working with pediatric patients benefit from understanding cognitive development processes of the child in order to more effectively communicate with

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TABLE 1-2: READS EXAMPLES IN PEDIATRIC PATIENTS

Principle	Example of the pharmacist's response
Roll with resistance	"You worry that your friends would judge you for using insulin. I can see how that would be difficult. It sounds like if your friends understood the importance of your medication, they would understand."
Express empathy	"You sound frustrated—not just to have to follow a special diet but also to have to inject yourself in front of your friends. It seems that you feel like you don't want to seem different."
Avoid argumentation	"It sounds like a lot to do to manage your diabetes. I would hate to see that you experience any complications because your sugars are out of control. Can we talk about some ways you can enjoy time with your friends without putting your health at risk?"
Develop discrepancy	"On the one hand, you feel embarrassed and different by having to use your insulin, but on the other hand, by properly managing your diabetes every day, you can prevent yourself from complications or hospitalizations and can still hang out with your friends."
Support self-efficacy	"It's a great start that you recognize the dietary restrictions and importance of insulin for your diabetes, even though they seem difficult or embarrassing."

them. Strategies for obtaining information and educating children will differ at various ages, and strategies will change as the level of maturity and understanding of the child increases.

From 2 to 7 years of age, children are in the preoperational cognitive developmental stage as described by Jean Piaget.[21] In this stage, Piaget describes children as unable to consider or comprehend any other point of view than their own and as such, thoughts and communication revolve around themselves. Also, children consider only the present time and place as they have no concept of time. They cannot understand cause and effect and do not have the ability to connect an action to their health status. Useful techniques for education at this age include hands-on activities. Patient education should focus on the most basic information such as when to take the medication and how the medication will taste.

Between the ages of 7 and 11 years, children are in the concrete operational cognitive phase, which is the beginning of the ability for a child to form logical and systematic thoughts.[21] Although they can understand and recognize concrete events, they still cannot process hypothetical situations. At this age, children begin to understand that diseases can be preventable. Medication education can be expanded to discuss adverse reactions. Motivational interviewing techniques are not useful at this age.

Beyond 12 years of age, a child (or now adolescent) enters the formal operational phase.[21] Adolescents are able to think in hypothetical and abstract terms but may require motivation and attention to do so.[20] By late adolescence, the understanding between cause and effect related to healthcare is possible, as they understand their behaviors can affect their healthcare. With the adolescent, it becomes important to develop trust before beginning with extensive education about medications and health. It is also important to direct questions and education to the adolescent and allow the adolescent to answer rather than the caregiver, who may just verify or supplement the answer with additional information. At times, the caregiver may be asked to leave the area in order to have a private discussion with the adolescent, particularly if issues such as contraception or sexual activity, alcohol, smoking, and drug use may be discussed. Adolescents may either be shy and embarrassed about such topics or give unexpected shocking answers.[21] It is also important to note that during this stage of development, adolescents are highly concerned with the acceptance of their peers; thus, adherence and health-related behaviors may be adversely affected. Regardless of the situation, the healthcare provider must remain professional

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and nonjudgmental. Motivational interviewing is beneficial and encouraged in this age group. However, it is important to keep realistic goals and an understanding of what barriers exist for the adolescent.

At any age, the patient or caregiver should be assessed for willingness or the ability to adhere to the proposed treatment regimen and comprehension should be evaluated. Comprehension strategies may require the caregiver or patient to verbalize the information they just received. Again, instead of asking close-ended questions, asking the patient to explain how to take the medication, what possible adverse reactions may occur, and what to do if they do occur is more useful to ascertain an understanding of the instructions. In order to improve understanding, it is critical to use layman terms and preferably in the patient's spoken language. If medication leaflets are provided, be certain they are at an appropriate reading level and specific to pediatric patients. The USP and other resources provide pictograms or pediatric-specific medication information that can be distributed.

CONCLUSION

Developing appropriate assessment skills in any setting requires practice. In this casebook, the reader will be provided the opportunity to evaluate pharmacologic regimens for common problems in the pediatric population. Additionally, most cases include a specific medication-related problem to be identified. This casebook, in addition to your didactic and experiential education (or residency and clinical experience), will allow you to self-assess your knowledge and skills in caring for pediatric patients.

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PART 1 | CASES

2.1 Anaphylaxis—Level 1 17
2.2 Nutrition—Level 2 20
2.3 Toxicology—Level 3 23

3.1 Patent Ductus Arteriosus—Level 1 27
3.2 Hypertension—Level 2 29
3.3 Congenital Heart Defects—Level 3 31

4.1 Asthma—Level 1 33
4.2 Cystic Fibrosis—Level 2 36
4.3 Respiratory Distress Syndrome and Bronchopulmonary Dysplasia—Level 3 40

5.1 Constipation—Level 1 43
5.2 Crohn Disease—Level 2 46

6.1 Nephrotic Syndrome—Level 2 49
6.2 Acute Kidney Injury—Level 3 52

7.1 Type 2 Diabetes Mellitus—Level 1 57
7.2 Hypothyroidism—Level 2 60
7.3 Diabetic Ketoacidosis—Level 3 66

8.1 Attention Deficit Hyperactivity Disorder—Level 1 69
8.2 Autism Spectrum Disorder—Level 2 72
8.3 Depression—Level 2 75
8.4 Cerebral Palsy—Level 3 78
8.5 Seizure Disorders—Level 3 81

9.1 Acute Otitis Media—Level 1 85
9.2 Skin and Skin Structure Infections—Level 1 87
9.3 Parasitic Infections—Level 2 90
9.4 Pneumonia—Level 2 92
9.5 Kawasaki Disease—Level 3 95
9.6 Human Immunodeficiency Virus Infection—Level 3 99
9.7 Meningitis—Level 3 102

10.1 Anemia—Level 1 105
10.2 Sickle Cell Disease—Level 2 108
10.3 Anticoagulation—Level 3 ... 111

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CASE 2.1
Anaphylaxis | Level 1

Nicole M. Even and Hanna Phan

LEARNING OBJECTIVES

1. Identify the signs and symptoms of an anaphylactic reaction.
2. Evaluate the anaphylactic reaction and an appropriate treatment regimen.
3. Recommend pharmacologic and nonpharmacologic therapy for an anaphylactic reaction.
4. Devise treatment goals and outcomes for each of the problems presented in the patient case.
5. Detect a medication-related problem requiring intervention in the patient case.

CHIEF COMPLAINT: Difficulty breathing

History of Present Illness: This is a 6-year-old female who initially presented to the emergency department with ear pain for the last 7 days. Her mother states she has been complaining that it really hurts to the point of crying; however, she won't let anyone touch it. While in the emergency department she is diagnosed with acute otitis media and is given a one-time dose of amoxicillin. Within 5 minutes of taking the antibiotic, she develops lip swelling, difficulty breathing, itching, hives, and flushing to the chest and upper extremities.

Review of Systems: Positive for swollen lips, positive labored breathing, pruritus, urticaria, and flushing to neck, chest, and upper extremities bilaterally

Past Medical History: Asthma and allergic rhinitis

Past Surgical History: None

Development History: No concerns

Social History: Lives with parents and an 8-year-old brother; attends 1st grade

Family History: None

Diet: Regular for age

Immunizations: Up to date, including the seasonal influenza vaccine

Allergies: Bee stings (per mother gets a rash around sting site)

PHYSICAL EXAM

BP 98/60 mm Hg | Pulse 160 beats per min | Temp 38.2ºC (oral)
RR 35 breaths per min | Wt 23 kg | Ht 118 cm | SpO_2 90% (RA)

General Appearance: Awake, alert, agitated, labored breathing, swollen lips, urticaria, flushing

Eyes: PERRLA, EOMs intact, conjunctiva clear

Ears: TM irritated with moderate bulging

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MEDICATION HISTORY

Medication	Sig	Start Date	End Date	Taking	Authorizing Provider
Albuterol MDI HFA 90 mcg/ inhalation	1–2 puffs q 4–6 hr prn SOB, wheeze, cough	2 yr ago		Yes	Dr. Greene
Fluticasone HFA 44 mcg/ inhalation	1 puff bid	2 yr ago		Yes	Dr. Greene
Children's multivitamin chewable tablet	1 tablet po daily	4 yr ago		Yes	Dr. Greene
Loratadine 5 mg/5 mL oral syrup	10 mg (10 mL) po daily	2 yr ago		Yes	Dr. Greene

SOB = shortness of breath.

Throat: Moist MM, throat normal, tongue mildly swollen, no posterior pharynx exudate, uvula swollen, able to swallow appropriately

Neck: Positive urticaria and flushing

Lungs: Negative wheeze, positive nasal flaring, positive dyspnea, positive tachypnea

Heart: Positive tachycardia, no murmur/ gallop/clicks

Abdomen: Soft, nontender, +BS, nondistended, no HSM

Musculoskeletal: Well perfused, capillary refill less than 2 seconds, no edema

Skin: Positive urticaria on neck, chest, and upper extremities bilaterally with flushing

Neurological: No gross deficits, strength 5 out of 5 upper and lower extremities, sensation intact, reflexes normal, interacting appropriately for age

LABORATORY DATA

BASIC METABOLIC PANEL		
Component	**Value**	**Range**
Glucose	75	60–110 mg/dL
BUN	8	8–19 mg/dL
Sodium	139	138–145 mmol/L
Potassium	3.8	3.4–5.2 mmol/L
Chloride	104	98–108 mmol/L
CO_2	23	20–28 mmol/L
Anion gap	12	6–16 mmol/L
Creatinine	0.4	0.2–0.7 mg/dL
Calcium	9.5	8.9–10.1 mg/dL
CALCULATED OSMOLALITY		
Calc osmo	285	285–295 mOsm/kg
CBC WITH DIFF		
WBC count	9.0	4.5–13.5 x 10^3/µL
RBC count	4.5	4.0–5.2 million/µL
Hemoglobin	13	11.5–15.5 g/dL
Hematocrit	39.5	35.0% to 45.0%
MCV	80	77.0–95.0 fL
MCH	30	25.0–33.0 pg
MCHC	33	32.0–35.0 g/dL
RDW	12	11.5% to 14.5%
Platelets	300	150–450 x 10^3/µL
MPV	10	9.4–12.4 fL
Neutrophils	70	34.0% to 71.1%
Immature granulocytes	0	0.0% to 0.5%
Neutrophils absolute	6.3	1.56–6.13 x 10^3/µL
Lymphocytes	25	19.3% to 51.7%
Lymphocytes absolute	2.25	1.18–3.74 x 10^3/µL
Monocytes	5	3.0% to 13.0%

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LABORATORY DATA (cont'd)

CBC WITH DIFF (cont'd)		
Monocytes absolute	0.45	0.24–0.86 x 10^3/μL
Eosinophils	0.7	0.7% to 5.8%
Eosinophils absolute	0.06	0.04–0.36 x 10^3/μL
Basophils	0.3	0.1% to 1.2%
Basophils absolute	0.027	0.01–0.08 x 10^3/μL

BUN = blood urea nitrogen; CBC = complete blood count; CO_2 = carbon dioxide; MCH = mean corpuscular hemoglobin; MCHC = mean corpuscular hemoglobin concentration; MCV = mean corpuscular volume; MPV = mean platelet volume; RBC = red blood cell; RDW = red cell distribution width; WBC = white blood cell.

DIAGNOSTIC TESTS

None performed

MEDICATIONS AT ADMISSION

Epinephrine 1:1000 0.23 mg IM once; may repeat in 5–15 min if needed

Diphenhydramine 12 mg IV q 6 hr prn pruritus

Albuterol 2.5 mg nebulized q 2 hr prn SOB

Fluticasone HFA 44 mcg/inhalation 1 puff inhaled bid

Loratadine 10 mg po daily

Children's multivitamin 1 tablet po daily

D5W–½NS at 65 mL/hr

0.5 L NC oxygen

Medications given immediately following reaction to amoxicillin

Epinephrine 0.23 mg IM × 1 dose

Diphenhydramine 12 mg IV × 1 dose

Albuterol MDI HFA 2 puffs

PROBLEM LIST

- ❑ Anaphylaxis
- ❑ Asthma
- ❑ Allergic rhinitis

SELF-ASSESSMENT QUESTIONS

1. What subjective and objective evidence supports the diagnosis of anaphylaxis?
2. Develop a plan for anaphylaxis for the patient upon discharge.
3. Assess the pharmacologic therapy for the patient's acute otitis media. Why is this therapy no longer appropriate? What medication recommendation can you make to treat this infection?
4. Devise the most pertinent patient education for the patient and caregiver.
5. What precautions need to be given as a result of this anaphylactic reaction regarding the patient's medical care communication in the future?

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CASE 2.2
Nutrition | Level 2

Sandra Benavides

LEARNING OBJECTIVES

1. Identify signs and symptoms of dehydration in an infant.
2. Calculate the required intravenous fluids (IVF) for rehydration in a patient.
3. Develop an initial parenteral nutrition (PN) regimen for a patient.
4. Develop the monitoring parameters during PN therapy.
5. Identify a medication-related problem in the patient case.

CHIEF COMPLAINT: Unable to tolerate gastrostomy tube (G-tube) feedings

History of Present Illness: A 7-month-old female infant with a history of trisomy 13 presented to the ED for evaluation of frequent chocking episodes. Two weeks ago, the mother took the infant to the GI physician to evaluate the episodes. The physician prescribed glycopyrrolate for increased secretions to decrease the chocking episodes. Mother reported the infant has been "gagging and dry heaving" with increased frequency over the past week. Per mother, the infant has not been tolerating her feedings through the G-tube, because she has had to stop the infusion on various occasions due to retching, vomiting, gagging, and irritability during feeding. It has gotten so bad that she has not provided feeds for last 24 hours. Mother reports the infant has a decreased number of wet diapers. She has had a bowel movement once in the last week, described as hard stool, which is not normal for her. Mother denies rash, fever, cough, rhinorrhea, diarrhea, or dysuria.

Review of Systems: Positive for vomiting, constipation, lethargy

Past Medical History:

- Trisomy 13 identified on amniocentesis at 6 months of pregnancy
- Cardiac abnormalities
- Bilateral cataracts
- Feeding difficulties (G-tube inserted at 1 month of life)

Birth History:

- Gestational age 37 weeks, 5 days
- Born to a 44-year-old, G3P1 mother
- Mother received prenatal care, positive for gestational diabetes controlled with diet
- Labor spontaneous, membranes ruptured at delivery, patient was born by urgent C-section for previous myomectomy in labor
- Patient remained in NICU for approximately 1 month, discharged at gestational age 43 weeks, 1 day

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MEDICATION HISTORY

Medication	Sig	Start Date	End Date	Taking	Authorizing Provider
Glycopyrrolate	0.7 mg G-tube 4 times daily prn drooling	1 wk ago		Yes	Dr. Schneider
Polyethylene glycol 3,350	8.5 g mixed with 8 oz formula q a.m.	3 mo ago		Yes	Dr. Schneider

Feeding: EleCare® 20 kcal/ounce, (56 cc/hr x 12 hours overnight; 90 cc q 3 hr during the day)

Past Surgical History: Face laceration from birth, repaired (1st day of life); gastrostomy with laparoscopic Nissen fundoplication (1 month of age secondary to poor feeding)

Social History: Lives with mother and father, no siblings, no pets in home

Family History: No family history of birth defects, genetic disorders, or developmental delay

Immunizations: Up to date per parents, including the first dose of the seasonal influenza vaccine 2 weeks ago

Allergies: NKDA

PHYSICAL EXAM

BP 94/68 mm Hg | Pulse 185 beats per min RR 46 breaths per min | Temp 36.3°C (axillary) | SpO_2 97% (room air) | Wt 7.2 kg (normal weight), weight today is 6.6 kg Length 67 cm

General Appearance: Lethargic, sleeping in mother's arms, limited responsiveness

Head: Normocephalic with obvious abnormality, sunken fontanel

Eyes: Normal lids, conjunctiva clear, visual acuity grossly intact, eyes sunken

ENT: Oropharynx normal, no mass, uvula midline, TM clear bilateral, nares clear, dry mucous membranes

Neck: Supple, no adenopathy

Heart: RRR, normal S1/S2, no murmurs/gallops/rubs, delayed capillary refill

Lungs: No respiratory distress, CTA bilaterally, no wheezes/rhonchi/rales, chest nontender to palpitation

Abdomen: Distended, tender, no masses, no HSM, no rebound, no guarding, G-tube 12 french 1.5 cm, site clear, no erythema or induration, no granulation tissue

GU: Deferred

Back: Normal inspection, full ROM without pain, tenderness, or spasm

Skin: Skin color, texture normal, increased skin turgor; no rashes or lesions, no petechiae

Neurological: No gross deficits, no meningismus

LABORATORY DATA

BASIC METABOLIC PANEL		
Component	**Value**	**Range**
Glucose	70	60–110 mg/dL
BUN	14	7–17 mg/dL
Sodium	138	137–145 mmol/L
Potassium	4.8	3.6–5.0 mmol/L
Chloride	100	98–107 mmol/L
CO_2	24	22–30 mmol/L
Creatinine	0.4	0.60–1.00 mg/dL

BUN = blood urea nitrogen; CO_2 = carbon dioxide.

PROBLEM LIST

- ❑ Dehydration
- ❑ PN with the same caloric intake as formula

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SELF-ASSESSMENT QUESTIONS

1. What is the subjective and objective evidence of dehydration in this patient?
2. Determine the severity of dehydration and devise a treatment for the patient in the case.
3. Develop an initial PN formula with the same caloric content as her current dietary intake for administration via a peripheral line.
4. Develop a monitoring plan for PN therapy.
5. List potential complications of PN.
6. Identify the medication-related problem and propose a solution.

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CASE 2.3
Toxicology | Level 3

Mary A. Babico and Hanna Phan

LEARNING OBJECTIVES

1. Identify the signs and symptoms of toxicities from polysubstance overdose.
2. Describe the mechanism of action of antidotes available for possible treatment in the patient case.
3. Devise treatment goals and outcomes for each of the identified healthcare problems presented in the patient case.
4. Identify any potential medication-related problems related to this patient's acute drug therapy.

CHIEF COMPLAINT: Unresponsive

History of Present Illness: Patient is a 16-year-old Caucasian female who was brought in to the emergency department by EMS. Patient was found unresponsive by her mother and sister this morning. Per report by patient's mother, patient has been stressed over the past 2–3 weeks with final exams and upcoming swim tournaments contributing to her anxiety. Patient arrived home last night tearful and angry after swim practice. She reportedly ate dinner, showered, and stayed in her room all night. Her mother called 911 after finding patient unresponsive in bed this morning. Ambulance arrival to home was around 07:20. Her mother looked through the medicine cabinet at home and found empty vials of the father's glipizide prescription, an empty vial of the patient's fluoxetine, and acetaminophen. It was estimated there were about 20 pills of glipizide, 15 capsules of fluoxetine, and less than 10 tablets of acetaminophen in their respective bottles. An empty box of Mucinex DM® was also found in patient's room. Time of ingestion is estimated between 18:30 and 23:00. The patient's sister later reported that patient has been using a friend's medication (name unknown) in the past few days to stay awake while studying.

Review of Systems: Unable to obtain given patient's altered mental status

Birth History: Not applicable

Past Medical History: Anxiety, depression, asthma

Past Surgical History: Unremarkable

Development History: Not applicable

Social History: Lives at home with both parents and younger sister; attends high school; active in sports

Family History: Father with type 2 diabetes mellitus

Diet: Regular for age

Immunizations: Up to date, including influenza vaccine this season

Allergies: NKDA

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MEDICATION HISTORY

Medication	Sig	Start Date	End Date	Taking	Authorizing Provider
Fluoxetine	20 mg po daily	3 weeks PTA		Yes	Dr. Smith
Loratadine	10 mg po daily	Chronic for 2 yr		Yes	Dr. Kennedy
Fluticasone	110 mcg 2 puffs bid	Chronic for 3 yr		Yes	Dr. Kennedy

PTA = prior to admission.

PHYSICAL EXAM

BP 116/74 mm Hg | Pulse 108 beats per min Temp 39°C (temporal) | RR 22 breaths per min | Wt 52 kg | Ht 153 cm | SpO_2 96% on room air | LMP 2 weeks ago

General Appearance: The patient is a well-developed, well-nourished 16-year-old female. The patient is occasionally responding to painful stimuli but is otherwise nonresponsive. She is in no respiratory distress.

Eyes: Pupils are 6 mm bilaterally and sluggishly reactive

Ears: No significant findings

Throat: Mucous membranes moist with significant amount of saliva

Neck: Supple

Lungs: Clear to auscultation bilaterally

Heart: Tachycardic, normal S1, S2; no murmurs appreciated

Abdomen: Soft, nondistended; positive bowel sounds

Musculoskeletal: Bilateral lower extremities in extension with toes pointed upward; rigid

Skin: Moist, warm; no rashes or lesions

Neurological: Patient's GCS is 6 upon arrival; does not open her eyes and has no verbal response; does withdraw to pain occasionally; ankle clonus measured at 12 Hz bilaterally

LABORATORY DATA

COMPLETE METABOLIC PANEL		
Component	**Value**	**Range**
Sodium	139	137–145 mmol/L
Potassium	3.1	3.6–5.0 mmol/L
Chloride	111	98–107 mmol/L
CO_2	16	22–30 mmol/L
BUN	9	7–17 mg/dL
Creatinine	0.8	0.7–1.2 mg/dL
Glucose	31	60–110 mg/dL
Calcium	9.8	8.4–10.2 mg/dL
Protein, total	7.6	6.3–8.2 g/dL
Albumin	4.2	3.5–5 g/dL
Bilirubin, total	0.4	0.2–1.3 mg/dL
ALP	83	38–126 g/dL
AST/SGOT	15	14–36 international units/L
ALT/SGPT	26	9–53 international units/L
Creatinine kinase	187	29–168 international units/L
Acetaminophen	12	10–30 mcg/mL
Salicylate	<20 (undetectable)	10–30 mcg/mL
COMPLETE BLOOD COUNT		
WBC count	12.8	4.5–11.0 x 10^3/µL
RBC count	4.33	4.30–5.10 million/µL
Hemoglobin	13.1	11.4–15.4 g/dL
Hematocrit	40.2	36.0% to 49.0%
MCV	93.7	78.0–98.0 fL
MCH	30.1	26.0–34.0 pg
MCHC	32.4	32.0–35.0 g/dL
MPV	7.7	9.4–12.4 fL
RDW	14.4	11.5% to 14.5%
Platelets	473	150–450 x 10^3/µL
ARTERIAL BLOOD GAS		
SaO_2	79.8	95% to 100%
pO_2	47	80–100 mm Hg

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LABORATORY DATA (cont'd)

ARTERIAL BLOOD GAS (cont'd)		
pCO_2	42	32–45 mm Hg
pH	7.28	7.32–7.42
Bicarbonate	21	22–26 mmol/L
Carboxyhemoglobin	2.4	0% to1.5%
Methemoglobin	1	0.4% to 1.5%
Hemoglobin total	13.9	11.5–15.5 g/dL
Base deficit	6.8	+/–2 mmol/L
Ionized calcium	1.21	1–1.18 mmol/L
Lactate	2.4	0.5–2.2 mmol/L
PREGNANCY, URINE		
Pregnancy test		Negative
TOXICOLOGY SCREEN, URINE		
Barbiturates		Undetected
Benzoylecgonine (cocaine)		Undetected
Benzodiazepine		Undetected
Amphetamine		Positive
D-9THC		Undetected
Opiate		Undetected
Phencyclidine		Undetected

ALP = alkaline phosphatase; ALT = alanine transaminase; AST = aspartate aminotransferase; BUN = blood urea nitrogen; CO_2 = carbon dioxide; D-9THC = delta-9-(trans)-tetrahydrocannabinol; MCH = mean corpuscular hemoglobin; MCHC = mean corpuscular hemoglobin concentration; MCV = mean corpuscular volume; MPV = mean platelet volume; pO_2 = partial pressure of oxygen; pCO_2 = partial pressure of carbon dioxide; RBC = red blood cell; RDW = red cell distribution width; SaO_2 = oxygen saturation of arterial blood; SGOT = serum glutamic oxaloacetic transaminase; SGPT = serum glutamic-pyruvic transaminase; WBC = white blood cell.

DIAGNOSTIC TESTS

CXR: Right upper lobe atelectasis

Head CT, Without Contrast: Unremarkable

ECG: Sinus tachycardia at a rate of 108 beats per min; normal axis; normal intervals; QRS 88 msec; PR 170 msec; QTc 432 msec with nonspecific ST and T-wave changes

Thin Layer Chromatography: Substances consistent with dextromethorphan, dextromethorphan metabolites, guaifenesin

MEDICATIONS AT ADMISSION

Normal saline 1 L bolus

Lorazepam 2 mg IV × 1 dose

Other drug therapy pending pharmacist's recommendations

PROBLEM LIST

- ❑ Serotonin syndrome secondary to dextromethorphan, fluoxetine, and dexamphetamine ingestion
- ❑ Glipizide toxicity due to intentional ingestion
- ❑ Dextromethorphan toxicity due to intentional ingestion
- ❑ Depression with suicidal ideation

SELF-ASSESSMENT QUESTIONS

1. What subjective and objective evidence supports the diagnosis of serotonin syndrome?
2. What subjective and objective evidence supports toxicities due to other ingested agents?
3. Devise a pharmacologic regimen for the treatment of this multiagent ingestion.
4. Assess the use of lorazepam in the treatment of this toxic ingestion.
5. Which monitoring parameters should be considered for the treatment of this multiagent poisoning? How often should they be checked?
6. Develop a patient education plan for this patient (and her caregiver/s) upon discharge.

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CASE 3.1
Patent Ductus Arteriosus | Level 1

Brady S. Moffett

LEARNING OBJECTIVES

1. Identify the presenting signs and symptoms of a patent ductus arteriosus (PDA) in an infant.
2. Recommend pharmacologic and nonpharmacologic therapy for PDA.
3. Devise treatment goals and outcomes for each of the identified healthcare problems presented in the patient case.

CHIEF COMPLAINT: Poor feeding and poor weight gain

History of Present Illness: The patient is a 6-day-old female born via vaginal delivery at 34 6/7 weeks gestation to a G1P1 mother. The patient was discharged home from the hospital on day 3 of life at a weight of 2.2 kg. Shortly after discharge, the mother noticed the infant was sweating while feeding and would tire prior to completing an entire feed. At the scheduled pediatrician visit earlier today, the patient had weight loss (current weight = 1.8 kg) and was tachypneic. The pediatrician noticed a murmur on auscultation and referred the patient to the emergency department. Subsequently, the patient was admitted to the Level II newborn nursery.

Review of Systems: Positive for tachypnea

Birth History: 34 6/7 weeks gestation

Past Medical History: None

Past Surgical History: None

Development History: None

Social History: None

Family History: Lives with mother and father

Diet: Breastfeeding

Immunizations: Up to date (hepatitis B at day of life one)

Allergies: None

Medication History: None

PHYSICAL EXAM

BP 78/42 mm Hg | Pulse 140 beats per min | Temp 98.8°F (rectal) | RR 52 breaths per min | Wt 1.8 kg | Ht 46 cm
SpO_2 98% on RA

General Appearance: Awake and alert infant

Eyes: PERRLA, EOMs intact, conjunctiva clear

Ears: TM clear bilaterally

Throat: Moist MM, throat normal, no posterior pharynx exudate

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Neck: No lymphadenopathy, supple with no meningismus, trachea midline

Lungs: CTA bilaterally with good aeration, no retractions, no nasal flaring

Heart: RRR, grade 3 murmur over left infraclavicular region, 2+ distal pulses

Abdomen: Soft, nontender, +BS, nondistended, no HSM

Musculoskeletal: Well perfused, capillary refill 3 to 4 seconds, patient appears mildly edematous

Skin: No rashes, no petechiae, no purpura

Neurological: No gross deficits, strength 5 out of 5 upper and lower extremity, sensation intact, reflexes normal, interacting appropriately for age

LABORATORY DATA

BASIC METABOLIC PANEL		
Component	**Value**	**Range**
Glucose	100	60–110 mg/dL
BUN	12	7–17 mg/dL
Sodium	143	137–145 mmol/L
Potassium	4.2	3.6–5.0 mmol/L
Chloride	100	98–107 mmol/L
CO_2	24	22–30 mmol/L
Anion gap	8	6–16 mmol/L
Creatinine	0.8	0.60–1.00 mg/dL
Calcium	9.2	8.4–10.2 mg/dL
CBC WITH DIFF		
WBC count	4.9	4.5–11.0 x 10^3/µL
RBC count	4.45	4.30–5.10 million/µL
Hemoglobin	12.2	11.4–15.4 g/dL
Hematocrit	44.0	36.0% to 49.0%
MCV	90.0	78.0–98.0 fL
MCH	30.0	26.0–34.0 pg
MCHC	33.4	32.0–35.0 g/dL
RDW	12.2	11.5% to 14.5%
Platelets	300	150–450 x 10^3/µL
MPV	11.4	9.4–12.4 fL
Neutrophils	42	34.0% to 71.1%
Immature granulocytes	0.1	0.0% to 0.5%
Neutrophils absolute	1.98	1.56–6.13 x 10^3/µL
Lymphocytes	30.1	19.3% to 51.7%
Lymphocytes absolute	1.33	1.18–3.74 x 10^3/µL
Monocytes	8.2	3.0% to 13.0%
Monocytes absolute	0.55	0.24–0.86 x 10^3/µL
Eosinophils	1.3	0.7% to 5.8%
Eosinophils absolute	0.22	0.04–0.36 x 10^3/µL
Basophils	0.98	0.1% to 1.2%
Basophils absolute	0.05	0.01–0.08 x 10^3/µL

BUN = blood urea nitrogen; CBC = complete blood count; CO_2 = carbon dioxide; MCH = mean corpuscular hemoglobin; MCHC = mean corpuscular hemoglobin concentration; MCV = mean corpuscular volume; MPV = mean platelet volume; RBC = red blood cell; RDW = red cell distribution width; WBC = white blood cell.

DIAGNOSTIC TESTS

Echocardiogram demonstrating a PDA with left to right shunting

MEDICATIONS AT ADMISSION

None

PROBLEM LIST

- ❑ Pulmonary overcirculation
- ❑ Heart failure
- ❑ PDA
- ❑ Pain management for minor procedures

SELF-ASSESSMENT QUESTIONS

1. What is the subjective and objective evidence for the diagnosis of PDA?
2. Assess the need for pharmacologic therapy for closure of a PDA.
3. Devise and assess a pharmacologic regimen for the treatment of pulmonary overcirculation and heart failure.
4. Determine the appropriate (if necessary) pharmacologic regimen for the treatment of PDA.
5. Devise pertinent education for the caregiver in regard to PDA.
6. Determine appropriate use of oral sucrose for pain management for minor procedures.

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CASE 3.2
Hypertension | Level 2

Brady S. Moffett

LEARNING OBJECTIVES

1. Identify the presenting signs and symptoms of hypertension in a pediatric patient.
2. Recommend initial pharmacologic and nonpharmacologic therapy for hypertension.
3. Recommend a pharmacotherapeutic monitoring strategy for a pediatric patient diagnosed with hypertension.
4. Devise treatment goals and outcomes for each of the identified healthcare problems presented in the patient case.

CHIEF COMPLAINT: None

History of Present Illness: A 14-year-old obese male presented to the pediatrician's office for a routine sports physical to play football. He had no health complaints. The patient's blood pressure was 145/95 mm Hg. He returned to the clinic 5 days later for a follow-up blood pressure check, which was 144/92 mm Hg. He was then referred to the cardiology outpatient hypertension clinic for further follow-up and sports clearance.

Birth History: Mother had gestational diabetes, patient was "large" at birth

Past Medical History: None; pediatrician has spoken to him and his mother about weight loss in the past

Past Surgical History: None

Development History: Unremarkable

Social History: Denies alcohol, tobacco, or illicit drug use

Family History: Lives with mother who works full time; obese; mother has been told she is at risk for diabetes

Diet: Consumes mostly fast food; drinks an "energy drink" 1–2 times per day

Immunizations: Up to date, including seasonal influenza vaccine

Allergies: None

Medication History: None

PHYSICAL EXAM

BP 145/95 mm Hg | Pulse 72 beats per min | Temp 36.4°C (oral)
RR 22 breaths per min | Wt 102 kg | Ht 178 cm
BMI 32.2 kg/m^2 (98th percentile) | SpO_2 98% (RA)

General Appearance: Awake, alert male obese child

Eyes: PERRLA, EOMs intact, conjunctiva clear

Ears: TM clear bilaterally

Throat: Moist MM, throat normal, no posterior pharynx exudate

Neck: No lymphadenopathy, supple with no meningismus, trachea midline

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Lungs: CTA bilaterally with good aeration, no retractions, no nasal flaring

Heart: RRR

Abdomen: Soft, nontender, +BS, non-distended, no HSM

Musculoskeletal: Well perfused, capillary refill 2 to 3 seconds

Skin: No rashes, no petechiae, no purpura

Neurological: No gross deficits, strength 5 out of 5 upper and lower extremity, sensation intact, reflexes normal, interacting appropriately for age

LABORATORY DATA

BASIC METABOLIC PANEL		
Component	**Value**	**Range**
Glucose	126	60–110 mg/dL
BUN	12	7–17 mg/dL
Sodium	141	137–145 mmol/L
Potassium	3.9	3.6–5.0 mmol/L
Chloride	104	98–107 mmol/L
CO_2	31	22–30 mmol/L
Creatinine	0.9	0.60–1.00 mg/dL

BUN = blood urea nitrogen; CO_2 = carbon dioxide.

DIAGNOSTIC TESTS

None

MEDICATIONS AT ADMISSION

None

PROBLEM LIST

- ❑ Hypertension
- ❑ Obesity
- ❑ Drug dosing in obesity

SELF-ASSESSMENT QUESTIONS

1. What is the subjective and objective evidence for the diagnosis of hypertension?
2. What are the criteria for a diagnosis of obesity in this patient?
3. Devise a nonpharmacologic regimen for the treatment of hypertension in this patient.
4. What other considerations should occur prior to initiating pharmacotherapy in this patient?
5. Devise a pharmacologic regimen for the treatment of hypertension in this patient.
6. Devise treatment goals and outcomes for each of the identified healthcare problems presented in the patient case.
7. Identify and manage issues in weight-based dosing associated with obese pediatric patients.

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CASE 3.3
Congenital Heart Defects | Level 3

Brady S. Moffett

LEARNING OBJECTIVES

1. Identify the presenting signs and symptoms of a Tetralogy of Fallot in a pediatric patient.
2. Recommend pharmacologic and nonpharmacologic therapy for Tetralogy of Fallot.
3. Devise treatment goals and outcomes for each of the identified healthcare problems presented in the patient case.

CHIEF COMPLAINT: Blue lips and tiring easily at school

History of Present Illness: The patient is a 7-year-old male with an unremarkable medical history prior to this visit. Over the past 6 months, his mother has noticed that he has been becoming more tired at school and has had difficulty in keeping up with peers at recess. She has also noticed that his lips and fingertips will occasionally appear blue or grey. He presents to the emergency department after having an episode while playing outside in which the patient states it became "really hard to breathe" and he had to sit down. He also states that his "heart hurt" during this episode, and the pain was relieved after he sat down and grabbed his knees.

Review of Systems: Positive for tachypnea and mild cyanosis of the lips

Birth History: Unremarkable

Past Medical History: None

Past Surgical History: None

Development History: Unremarkable

Social History: Lives with mother and father

Family History: Unremarkable

Diet: Regular for age

Immunizations: Up to date, including seasonal influenza vaccine

Allergies: NKDA

MEDICATION HISTORY

Multivitamin daily

PHYSICAL EXAM

BP 100/70 mm Hg | Pulse 80 beats per min | Temp 36.5°C (oral)
RR 38 breaths per min | Wt 35 kg | Ht 98 cm | SpO_2 89% (RA)

General Appearance: Awake, alert male child, with mild cyanosis of lips

Eyes: PERRLA, EOMs intact, conjunctiva clear

Ears: TM clear bilaterally

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Throat: Moist MM, throat normal, no posterior pharynx exudate

Neck: No lymphadenopathy, supple with no meningismus, trachea midline

Lungs: CTA bilaterally with good aeration, no retractions, no nasal flaring

Heart: RRR, III/VI murmur on the left upper sternal border

Abdomen: Soft, nontender, +BS, nondistended, no HSM

Musculoskeletal: Well perfused, mildly cyanotic, capillary refill 2 to 3 seconds

Skin: No rashes, no petechiae, no purpura

Neurological: No gross deficits, strength 5 out of 5 upper and lower extremity, sensation intact, reflexes normal, interacting appropriately for age

LABORATORY DATA

BASIC METABOLIC PANEL		
Component	**Value**	**Range**
Glucose	82	60–110 mg/dL
BUN	12	7–17 mg/dL
Sodium	138	137–145 mmol/L
Potassium	3.8	3.6–5.0 mmol/L
Chloride	100	98–107 mmol/L
CO_2	26	22–30 mmol/L
Creatinine	0.7	0.60–1.00 mg/dL
Calcium	9.2	8.4–10.2 mg/dL
CBC WITH DIFF		
WBC count	4.8	4.5–11.0 x 10^3/µL
RBC count	4.9	4.30–5.10 million/µL
Hemoglobin	12.2	11.4–15.4 g/dL
Hematocrit	37.0	36.0% to 49.0%
MCV	88.8	78.0–98.0 fL
MCH	28.8	26.0–34.0 pg
MCHC	33.4	32.0–35.0 g/dL
RDW	12.2	11.5% to 14.5%
Platelets	180	150–450 x 10^3/µL
MPV	10.1	9.4–12.4 fL
Neutrophils	38.8	34.0% to 71.1%
Immature granulocytes	0.1	0.0% to 0.5%
Neutrophils absolute	1.74	1.56–6.13 x 10^3/µL
Lymphocytes	22.2	19.3% to 51.7%
Lymphocytes absolute	1.96	1.18–3.74 x 10^3/µL
Monocytes	5.3	3.0% to 13.0%
Monocytes absolute	0.33	0.24–0.86 x 10^3/µL
Eosinophils	2.9	0.7% to 5.8%
Eosinophils absolute	0.21	0.04–0.36 x 10^3/µL
Basophils	0.8	0.1% to 1.2%
Basophils absolute	0.02	0.01–0.08 x 10^3/µL

BUN = blood urea nitrogen; CBC = complete blood count; CO_2 = carbon dioxide; MCH = mean corpuscular hemoglobin; MCHC = mean corpuscular hemoglobin concentration; MCV = mean corpuscular volume; MPV = mean platelet volume; RBC = red blood cell; RDW = red cell distribution width; WBC = white blood cell.

DIAGNOSTIC TESTS

Echocardiogram demonstrating pulmonary stenosis, a ventricular septal defect with occasional right-to-left shunting, a thickened right ventricle, and an overriding aorta

MEDICATIONS AT ADMISSION

None

PROBLEM LIST

- ❑ Tetralogy of Fallot
- ❑ Hypercyanotic spells

SELF-ASSESSMENT QUESTIONS

1. What is the subjective and objective evidence for the diagnosis of Tetralogy of Fallot?
2. Devise a pharmacologic regimen for prevention of a hypercyanotic spell secondary to Tetralogy of Fallot.
3. Devise a pharmacologic regimen for the treatment of a life-threatening hypercyanotic spell secondary to Tetralogy of Fallot.
4. Devise pertinent patient and caregiver education for the prophylaxis and treatment of hypercyanotic spell in Tetralogy of Fallot.
5. Determine the most appropriate medication dosage form for propranolol in the prophylaxis of hypercyanotic spells in Tetralogy of Fallot.

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CASE 4.1
Asthma | Level 1

Manar O. Lashkar and Hanna Phan

LEARNING OBJECTIVES

1. Identify the presenting signs and symptoms of asthma in a pediatric patient.
2. Identify the criteria for intensive care unit admission.
3. Evaluate the stage of asthma control for a pediatric patient and construct an appropriate treatment.
4. Recommend pharmacologic therapy for moderate persistent asthma.
5. Provide treatment goals and outcomes for each of the identified healthcare problems presented in the case.
6. Detect a medication-related problem requiring intervention in the patient case.

CHIEF COMPLAINT: Unable to breathe

History of Present Illness: This is a 7-year-old African American male with asthma presenting to the emergency department (ED) this morning after missing 2 days from school this week due to shortness of breath that was not alleviated with rescue inhaler use every 4 hours. Since school started 3 months ago, he has missed school a day or two in each month, and he used about two albuterol canisters in the past month. He has not been able to participate in gym class as he coughs during the school day; but, according to his mother, "this is normal, he has always had breathing problems, it was just worse than normal this time." He wakes up at night coughing and asks for his inhaler almost every other day, but it has been daily in the past week. He was admitted to the hospital last fall for similar symptoms, was started on a controller inhaler at that time, and diagnosed with asthma. His mother states that she has not followed up with his pediatrician regularly for his asthma; he was seen once in the last year.According to mother, the patient has visited urgent care and the ED for asthma symptoms two times this year, one of which he was prescribed an oral steroid course for 5 days and the other he was admitted to the pediatric intensive care unit (PICU) for 2 days (not intubated). Today, in the ED, the patient's symptoms did not improve with albuterol and ipratropium, and the patient continued with severe symptoms at rest including dyspnea to the point that he is unable to speak full sentences. It was determined to admit the patient to the PICU and is in transit to unit.

Review of Systems: Positive for inspiratory and expiratory wheezing, shortness of breath, runny nose, watery eyes, and dyspnea (in which he is unable to speak full sentences)

Past Medical History: Asthma (diagnosed 1 year ago), allergic rhinitis, and attention deficit hyperactivity disorder (ADHD)

Past Surgical History: None

Development History: No concerns

Social History: Lives with parents and a sister and attends 1st grade

Family History: Mother with history of childhood asthma, father with history of seasonal allergies

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MEDICATION HISTORY

Medication	Sig	Start Date	End Date	Taking	Authorizing Provider
Fluticasone 44 mcg HFA MDI inhaler	2 puffs bid with spacer	10/2012		Yes	Dr. Smith
Albuterol 90 mcg HFA MDI inhaler	2–4 puffs q 6 hr prn wheeze or cough	10/2012		Yes	Dr. Smith
Loratadine tablet	5 mg po daily	10/2012		Yes	Dr. Smith
Methylphenidate LA tablet	30 mg po daily	08/2012		Yes	Dr. Smith

Diet: Regular for diet

Immunizations: Last vaccinations were prior to kindergarten admission last year; no age-scheduled or seasonal vaccination since then

Allergies: Seasonal allergies, NKDA

PHYSICAL EXAM

BP 100/60 mm Hg | Pulse 140 beats per min
Temp 36.8°C (oral) | RR 42 breaths per min
Wt 30 kg | Ht 120 cm | SpO_2 85% on RA, PEF personal best 200 L/min, current PEF 70 L/min

General Appearance: Awake, agitated, in discomfort, and talks in words

Eyes: PERRLA, EOMs intact, conjunctivitis

Ears: TM clear bilaterally

Throat: Moist MM, throat normal, no posterior pharynx exudate

Neck: No lymphadenopathy, supple with no meningismus, trachea midline

Lungs: Increased anterior-posterior diameter of the chest, hyperinflation with nasal flaring

Heart: RRR, no murmur/gallop/clicks, 2+ distal pulses

Abdomen: Soft, nontender, +BS, nondistended, no HSM

Musculoskeletal: Well perfused, capillary refill less than 2 seconds, no edema

Skin: No rashes, no petechiae, no purpura

Neurological: No gross deficits, strength 5 out of 5 upper and lower extremity, sensation intact, reflexes normal, interacting appropriately for age

LABORATORY DATA

BASIC METABOLIC PANEL		
Component	**Value**	**Range**
Glucose	115	60–110 mg/dL
BUN	10	8–19 mg/dL
Sodium	140	138–145 mmol/L
Potassium	3	3.4–6 mmol/L
Chloride	100	98–107 mmol/L
CO_2	28	20–28 mmol/L
Anion gap	12	6–16 mmol/L
Creatinine	0.6	0.2–0.7 mg/dL
Calcium	9	8.9–10.1 mg/dL
OSMOLALITY		
Serum osmolality	290	285–295 mOsm/kg
CBC WITH DIFF		
WBC count	7.0	4.5–13.5 x 10^3/µL
RBC count	4.8	4–5.2 million/µL
Hemoglobin	11.6	11.5–15.5 g/dL
Hematocrit	35	35% to 45%
MCV	78	77–95 fL
MCH	26	25–33 pg
MCHC	33	32–35 g/dL
RDW	11.6	11.5% to 4.5%
Platelets	200	150–450 x 10^3/µL
MPV	9.6	9.4–12.4 fL
Neutrophils	50	34.0% to 71.1%
Immature granulocytes	0	0.0% to 0.5%
Neutrophils absolute	3.5	1.56–6.13 x 10^3/µL
Lymphocytes	30	19.3% to 51.7%
Lymphocytes absolute	2.1	1.18–3.74 x 10^3/µL
Monocytes	10	3.0% to 13.0%
Monocytes absolute	0.7	0.24–0.86 x 10^3/µL
Eosinophils	1	0.7% to 5.8%

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LABORATORY DATA (cont'd)

CBC WITH DIFF (cont'd)		
Eosinophils absolute	0.07	0.04–0.36 x 10^3/μL
Basophils	1	0.1% to 1.2%
Basophils absolute	0.07	0.01–0.08 x 10^3/μL

BUN = blood urea nitrogen; CBC = complete blood count; CO_2 = carbon dioxide; MCH = mean corpuscular hemoglobin; MCHC = mean corpuscular hemoglobin concentration; MCV = mean corpuscular volume; MPV = mean platelet volume; RBC = red blood cell; RDW = red cell distribution width; WBC = white blood cell.

DIAGNOSTIC TESTS

Chest radiograph (report): Increased bronchial wall markings, flattening of diaphragm, and hyperinflation

MEDICATIONS AT ADMISSION

Albuterol 15 mg/hr continuous nebulizer

Prednisolone liquid 15 mg po × 1 dose (given in ED; however, patient experienced emesis soon after dose due to severe cough)

D5W–½NS at 80 mL/hr

Loratadine 5 mg po daily (held as patient is npo)

PROBLEM LIST

- ❑ Severe asthma exacerbation
- ❑ Chronic asthma, uncontrolled
- ❑ Attention deficit hyperactivity disorder
- ❑ Possible medication nonadherence

SELF-ASSESSMENT QUESTIONS

1. What subjective and objective evidence exists in the patient to support a diagnosis of severe asthma exacerbation?
2. List risk factors that necessitate intensive care unit admission in a patient with asthma exacerbation. Is this patient considered high risk with regard to his asthma?
3. Determine the classification of asthma control for the patient. Which components of control did the patient present with to support this classification?
4. Assess the patient's current acute pharmacologic therapy for asthma.
5. Identify a potential medication-related adverse reaction associated with asthma exacerbation therapy and its management.
6. Devise a discharge pharmacologic regimen for his asthma and other conditions.

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CASE 4.2
Cystic Fibrosis | Level 2

Hanna Phan

LEARNING OBJECTIVES

1. Identify the presenting signs and symptoms of cystic fibrosis (CF) acute pulmonary exacerbation in a pediatric patient.
2. Apply pharmacokinetic differences noted in pediatric patients with CF in drug therapy selection and dosing.
3. Recommend a pharmacologic regimen for treatment of an acute pulmonary exacerbation in a pediatric patient with CF.
4. Develop a discharge plan for a pediatric patient with CF including patient/caregiver education.
5. Identify a medication-related problem and develop a plan for resolution of the problem.

CHIEF COMPLAINT: "I have been coughing more often than usual and coughing up more 'green-yellow stuff.' I don't want to be sick right before my birthday."

History of Present Illness: An 11-year-old boy came to the CF clinic with his mother for a routine quarterly visit and reported a 1-week history of increased cough and sputum production. The patient stated he has been more tired in the last few days and had to miss school the last 2 days. His mother stated the household has been fighting off a "cold" for the past few weeks, and her son was the last to "catch it." She also reported the patient has had less of an appetite, has been eating fewer snacks, and has difficulty finishing any of his meals while he has not been feeling well. The patient reports no changes in bowel movements with the exception of occasionally floating with greasy stools, and the last incident about 3 days ago. His mother started increased airway clearance therapy with his vest for the past 3 days, which has increased from once daily to twice daily, without much improvement in his symptoms. He denied fever, vomiting, hemoptysis, and abdominal pain or discomfort. The attending physician asks the pharmacist to develop a pharmacologic regimen for his admission for CF acute pulmonary exacerbation.

Review of Systems: Positive for increased cough and sputum production and respiratory distress

Birth History: Born at 38 weeks via NSVD, no complications except meconium ileus that initiated an evaluation for CF

Past Medical History: Patient was diagnosed with CF at 1 month of age through combination of newborn screen, workup, and borderline sweat test, followed with confirmation by genotyping (delta-F508 homozygous). Pancreatic insufficiency secondary to CF; asthma and allergic rhinitis was diagnosed at age 4 years; GERD was diagnosed 3 years ago; and ADHD was recently diagnosed 1 month ago. Patient's baseline FEV_1% predicted is 98. MRSA and *P. aeruginosa* has grown from sputum cultures in the past 12 months.

Past Surgical History: None

Development History: In kindergarten, no history of developmental delay

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MEDICATION HISTORY

Medication	Sig	Start Date	End Date	Taking	Authorizing Provider
Albuterol 0.083% inhalation solution	2.5 mg nebulized bid	Since diagnosis		Currently taking	Dr. Lee
Dornase alfa inhalation solution	2.5 mg nebulized daily	Since 3 yr of age		Nonadherent	Dr. Lee
Tobi Podhaler®	112 mg (4 of the 28 mg capsules) inhaled bid—28 days on/off	12/01/2013		Currently taking	Dr. Lee
Lansoprazole capsule	15 mg po daily	02/01/2011		Currently taking	Dr. Lee
Creon® 12,000 capsules	5 caps po with meals, 3 caps po with snacks	Dose adjusted 6 mo ago		Currently taking	Dr. Lee
Cetirizine tablet	10 mg po daily	06/01/2010		Currently taking	Dr. Smith
Fluticasone 44 mcg HFA MDI inhaler	2 puffs inhaled with spacer bid	03/01/2010		Nonadherent	Dr. Lee
Hypertonic saline 7% inhalation solution	4 mL nebulized daily	06/01/2010		Nonadherent	Dr. Lee
AquADEK® MVI capsule	1 cap po daily	Since age 9 yr		Currently taking	Dr. Lee
Azithromycin tablets	250 mg po MWF	Since age 6 yr		Currently taking	Dr. Lee
Ibuprofen tablets	400 mg po bid	Dose last adjusted 6 mo ago		Currently taking	Dr. Lee
Methylphenidate LA capsules	40 mg po daily	1 mo ago		Currently taking	Dr. Smith

Social History: Lives with his mother, father, and his 14-year-old sister; pets at home include a golden retriever (Sunny)

Family History: 2nd cousin on paternal side has CF; maternal history of asthma

Diet: High-calorie diet, BOOST® Kids (two cans/day) supplements for additional calorie intake

Immunizations: Family previously declined all immunizations, but patient caught up with routine childhood schedule in the last 3 months, except annual influenza

Allergies: Sulfamethoxazole–trimethoprim (hives), penicillin (rash, tolerates cephalosporins)

PHYSICAL EXAM

BP 100/60 mm Hg | Pulse 90 beats per min
Temp 37.1°C (temporal) | RR 24 breaths per min
Wt 26 kg (lost 1.5 kg from 4 months ago)
Ht 132 cm | SpO_2 87% on room air

General Appearance: Alert, actively coughing throughout exam

HEENT: Normocephalic, PERRLA, EOMI, moist oral mucosa, nasal polyp on right

Lungs: Crackles and rhonchi on RLL and LUL, no retractions or wheeze

Heart: RRR, no murmurs or irregularities noted

Abdomen: Soft, nontender, nondistended with positive bowel sounds; no hepatosplenomegaly

Musculoskeletal: Minor clubbing of upper extremities

Skin: No exudates or erythema

Neurological: Neurologically appropriate; nonfocal exam; cranial nerves II–XII intact

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LABORATORY DATA

COMPREHENSIVE METABOLIC PANEL		
Component	**Value**	**Range**
Glucose	95	60–110 mg/dL
BUN	14	7–17 mg/dL
Sodium	140	137–145 mmol/L
Potassium	4.2	3.6–5.0 mmol/L
Chloride	107	98–107 mmol/L
CO_2	22	22–30 mmol/L
Anion gap	11	6–16 mmol/L
Creatinine	0.7	0.60–1.00 mg/dL
Calcium	9.1	8.4–10.2 mg/dL
Albumin	3.3	3.7–4.8 g/dL
Protein	6.9	6.4–8.5 g/dL
AST (SGOT)	25	5–34 international units/L
ALT (SPGT)	16	9–69 international units/L
ALP	185	92–549 international units/L
Total bilirubin	0.2	0.2–1.1 mg/dL
CBC WITH DIFF		
WBC count	12.0	4.5–11.0 x 10^3/µL
RBC count	4.51	4.30–5.10 million/µL
Hemoglobin	12	11.4–15.4 g/dL
Hematocrit	36	36.0% to 49.0%
MCV	79.8	78.0–98.0 fL
MCH	25.2	26.0–34.0 pg
MCHC	31.6	32.0–35.0 g/dL
RDW	14.5	11.5% to 14.5%
Platelets	320	150–450 x 10^3/µL
MPV	10	9.4–12.4 fL
Neutrophils	59	34.0% to 71.1%
Immature granulocytes	0.0	0.0% to 0.5%
Neutrophils absolute	29.2	1.56–6.13 x 10^3/µL
Lymphocytes	29.2	19.3% to 51.7%
Lymphocytes absolute	3.30	1.18–3.74 x 10^3/µL
Monocytes	8.2	3.0% to 13.0%
Monocytes absolute	0.90	0.24–0.86 x 10^3/µL
Eosinophils	3.3	0.7% to 5.8%
Eosinophils absolute	0.40	0.04–0.36 x 10^3/µL
Basophils	0.3	0.1% to 1.2%
Basophils absolute	0.00	0.01–0.08 x 10^3/µL
IBUPROFEN SERUM LEVELS		
1 hr postdose	20	50–100 mcg/mL (for CF)
2 hr postdose	30	50–100 mcg/mL (for CF)
3 hr postdose	45	50–100 mcg/mL (for CF)

VITAMIN SERUM LEVELS		
Vitamin A (retinol)	0.15	0.2–0.5 mg/L
Vitamin D 25-OH total	20	25–80 ng/mL
Vitamin E (alpha tocopherol)	4.5	5.5–9 mg/L
COAGULATION—GENERAL		
Prothrombin time	11.5	10–12 sec
INR	1.1	0.95–1.2

ALP = alkaline phosphatase; ALT = alanine transaminase; AST = aspartate aminotransferase; BUN = blood urea nitrogen; CBC = complete blood count; CF = cystic fibrosis ; CO_2 = carbon dioxide; INR = international normalized ratio; MCH = mean corpuscular hemoglobin; MCHC = mean corpuscular hemoglobin concentration; MCV = mean corpuscular volume; MPV = mean platelet volume; RBC = red blood cell; RDW = red cell distribution width; WBC = white blood cell.

CF Sputum Culture

- Fungal—negative, final
- Acid-fast bacilli—negative, final
- Gram stain—1+ WBC, moderate gram-positive cocci, moderate gram-negative rods

Moderate growth methicillin-resistant *S. aureus*

Antibiotic	Susceptibility (MIC)
Clindamycin	R
Erythromycin	R (>8)
Gentamicin	S (≤0.05)
Linezolid	S (2)
Oxacillin	R (>4)
Tetracycline	S (≤1)
Trimethoprim–sulfamethoxazole	S (≤0.5/9.5)
Vancomycin	S (≤0.5)

MIC = minimal inhibitory concentration; R = resistant; S = susceptible.

Moderate growth, *P. aeruginosa*

Antibiotic	Susceptibility (MIC)
Amikacin	I (32)
Aztreonam	I (16)
Cefepime	S (4)
Ciprofloxacin	S (1)
Gentamicin	R (>4)
Meropenem	I (8)
Piperacillin–tazobactam	S (≤8)
Tobramycin	S (2)

MIC = minimal inhibitory concentration; I = intermediate; R = resistant; S = susceptible.

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DIAGNOSTIC TESTS

Spirometry (current):

$FEF_{25–75}$ % predicted = 66

$FEF_{25–75}$ = 1.52 L/sec

FEV_1 = 1.65 L

FEV_1 % predicted = 82

FEV_1/FVC = 0.79

FVC = 2.09 L

FVC % predicted = 89

MEDICATIONS AT ADMISSION

To be determined by pharmacist

PROBLEM LIST

- ❑ CF acute pulmonary exacerbation
- ❑ Pancreatic insufficiency, poor weight gain
- ❑ ADHD
- ❑ Asthma and allergic rhinitis

SELF-ASSESSMENT QUESTIONS

1. What is the subjective and objective evidence for the diagnosis of CF acute pulmonary exacerbation in this patient?
2. Describe recommended nonpharmacologic therapy as part of treatment for this patient's CF acute pulmonary exacerbation.
3. Develop a pharmacologic regimen for the treatment of CF acute pulmonary exacerbation for this patient, including goals of therapy and monitoring parameters.
4. Evaluate the current regimen for chronic CF in this patient and recommend any necessary changes.
5. Identify other medication-related problem(s) and develop a solution.

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CASE 4.3
Respiratory Distress Syndrome and Bronchopulmonary Dysplasia | Level 3

Hanna Phan

LEARNING OBJECTIVES

1. Identify the presenting signs and symptoms of respiratory distress syndrome (RDS) and bronchopulmonary dysplasia (BPD) in a premature neonate.
2. Determine risk factors and approaches to prevent RDS and BPD in premature neonates.
3. Recommend treatment regimens for RDS and BPD in a premature neonate.
4. Develop a discharge plan for a patient with BPD including caregiver education.
5. Identify a medication-related problem and develop a plan for resolution of the problem.

CHIEF COMPLAINT: Failed attempts to wean ventilator settings and increased respiratory symptoms

History of Present Illness: Patient is a 5-week-old premature male in the NICU with continued respiratory distress. The patient has required mechanical ventilation since birth due to RDS secondary to prematurity. He has failed attempts to wean the ventilator to lower FiO_2 settings and continues to have episodes of increased work of breathing, tachypnea, and retractions despite the use of albuterol. The team would like to transition the patient to continuous positive airway pressure (CPAP) for supplemental oxygen support. The patient recently had increased respiratory secretions for which glycopyrrolate was started 3 days ago. The patient appears to be increasingly edematous over the past week and has decreased urine output.

Birth History: Born at 26 weeks gestation to a G2P2 mother, RDS at birth, and received three doses of poractant alfa 2 mL intratracheal for one dose, then 1 mL for two doses; mother was GBS (-) and did not receive antenatal steroids prior to delivery (vaginal delivery); birth weight 800 g; APGAR at 1, 5, 10, and 15 minutes were 3, 5, 6, and 7

Past Medical History: Neonatal RDS at birth treated with surfactant, mechanical ventilation since birth, history of PDA treated with ibuprofen, resolved; completed 4-week course of vitamin A therapy for BPD prevention

Past Surgical History: None

Development History: Patient is premature, reflexes normal for age

Social History: Mother—history of illicit drug abuse but was undergoing rehabilitation prior to pregnancy, no noted drug use during this pregnancy; father—unknown

Family History: Maternal side of family affected by asthma and depression (MGM); unknown history from paternal side

Diet: Similac NeoSure® 22 kcal/30 mL, receiving full enteral feeds at 270 mL (15 mL/hr) as continuous feeds (132 kcal/kg/day)

Immunizations: Awaiting weight to be 2 kg before administering first dose of hepatitis B vaccine

Allergies: NKDA

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MEDICATION HISTORY

Medication	Sig	Start Date	End Date	Taking	Authorizing Provider
Poly-Vi-Sol®	1 mL po daily	2 wk ago		Currently taking	Dr. Smith
Glycopyrrolate solution (0.2 mg/mL)	0.15 mg po 4 times daily	3 days ago		Currently taking	Dr. Newbie
Albuterol sulfate 0.083%	1.25 mg nebulized q 6 hr	3 wk ago		Currently taking	Dr. Smith
Dexamethasone	0.38 mg po bid	1 day ago		Currently taking	Dr. Newbie

PHYSICAL EXAM

BP 55/28 mm Hg | Pulse 125 beats per min
Temp 37°C | 35 breaths per min | Wt 1.5 kg
Ht 42 cm | SpO_2 83%

Ventilator settings: SIMV-PC, PEEP 7 cm H_2O, PS 10 cm H_2O, PC 17, inspiratory time 0.5 sec, RR 20 breaths per min, FiO_2 50%

General Appearance: Alert, increased work of breathing despite ventilator, appears edematous

Eyes: PERRLA, clear

Ears: TM clear, no issues

Throat: Defer, intubated

Neck: No issues

Lungs: Course rhonchi noted, diffuse rales and wheeze on auscultation

Heart: History of PDA, now resolved; most recent ECHO without evidence of cardiac hypertrophy or pulmonary HTN; murmur has been heard intermittently on exam

Abdomen: Soft, nontender, nondistended; no masses felt; no hepatosplenomegaly; positive bowel sounds

Musculoskeletal: Warm and well-perfused; no cyanosis; capillary refill less than 2 seconds; edema noted in upper and lower extremities

Skin: No rashes, no petechiae, no purpura

Neurological: Alert, good tone; reflexes appropriate for age

LABORATORY DATA

BASIC METABOLIC PANEL		
Component	**Value**	**Range**
Glucose	70	60–110 mg/dL
BUN	5	7–17 mg/dL
Sodium	138	137–145 mmol/L
Potassium	3.8	3.6–5.0 mmol/L
Chloride	102	98–107 mmol/L
CO_2	23	22–30 mmol/L
Anion gap	13	6–16 mmol/L
Creatinine	0.4	0.60–1.00 mg/dL
Calcium	8.6	8.4–10.2 mg/dL
CBC		
WBC count	8.0	5.0–19.0 x 10^3/µL
RBC count	3.5	3–5.4 million/µL
Hemoglobin	11	10–18 g/dL
Hematocrit	31	31% to 55%
MCV	90	85–123 fL
MCH	30	28–40 pg
MCHC	32	29–37 g/dL
Platelets	200	150–450 x 10^3/µL
CAPILLARY BLOOD GAS		
pO_2	35	70–95 mm Hg
pCO_2	42	35–45 mm Hg
pH	7.35	7.35–7.45
Calculated bicarbonate	23.2	16–23 mmol/L
Base deficit	2.4	0–2 mmol/L

BUN = blood urea nitrogen; CBC = complete blood count; CO_2 = carbon dioxide; MCH = mean corpuscular hemoglobin; MCHC = mean corpuscular hemoglobin concentration; MCV = mean corpuscular volume; pO_2 = partial pressure of oxygen; pCO_2 = partial pressure of carbon dioxide; RBC = red blood cell; WBC = white blood cell.

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DIAGNOSTIC TESTS

Chest radiograph notes diffuse haziness throughout, hypoinflation on RLL

PROBLEM LIST

- ❑ BPD secondary to neonatal RDS and mechanical ventilation
- ❑ Prematurity

SELF-ASSESSMENT QUESTIONS

1. What is the subjective and objective evidence for the diagnosis of BPD in this patient?
2. Describe the risk factors for, clinical presentation of, and recommended treatment for neonatal RDS.
3. Discuss approaches in the prevention of BPD in premature neonates.
4. Develop a pharmacologic regimen for the treatment of BPD for this patient. Why are some previously used medications, such as systemic corticosteroids, no longer recommended?
5. Identify the medication-related problem(s) and develop a solution.
6. When is a patient with BPD considered ready for discharge? Describe what should be included in caregiver education for discharge.

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CASE 5.1
Constipation | Level 1

Jeremy S. Stultz and Milap C. Nahata

LEARNING OBJECTIVES

1. Identify the presenting signs and symptoms of constipation in children.
2. Determine when constipation in children is treatable with over-the-counter (OTC) medications.
3. Recommend OTC nonpharmacologic and pharmacologic treatment options for constipation.
4. Identify appropriate patient counseling points.
5. Detect a medication-related problem requiring intervention.

CHIEF COMPLAINT: Constipation for the past week with belly pain being reported over the past 2 days

History of Present Illness: A mother presents to the clinic located near your community pharmacy with her 3-year-old son. The mother describes her son as having difficulty "pooping" for the past week. Normally he has at least one bowel movement daily, but he has only had two small bowel movements over the past week. He has also been more fidgety and standing on his tiptoes a lot for the past 3 to 4 days. He was potty trained over the past 6 months (finishing 2 months ago) and started pre-kindergarten last month. Per his mother, he had a "stomach bug" a few weeks ago that caused significant diarrhea. During his illness, he had an "accident" where he did not get to the bathroom in time and was sent home from school. After a brief visit at an urgent care clinic and spending a few days at home, he improved and returned to pre-kindergarten last week. The mother has talked with her son about his bowel movements; originally the child stated he did not have to "poop" and now he states he can't. The nurse from the clinic called the pharmacy and said she was sending the mother and son to the pharmacy to discuss OTC treatment options for the patient's constipation.

Review of Systems: Positive for gastrointestinal pain and decreased stool production

Birth History: Previously born at full term, vaginal delivery; mother was G1P1 at the time

Past Medical History: Mild, well controlled asthma (mother reports worse in fall and spring), one hospitalization for anaphylaxis associated with a bee sting last year

Past Surgical History: None

Development History: Reaching appropriate milestones

Social History: Recently began pre-kindergarten

Family History: Lives at home with parents, older sister (7), and younger brother (1)

Diet: Very picky eater, has trouble eating fruits, vegetables, and cereal

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MEDICATION HISTORY

Medication	Sig	Start Date	End Date	Taking	Authorizing Provider
Ibuprofen 100 mg/5 mL suspension	7.5 mL po 4 times daily	Yesterday		Yes	OTC per mother
Albuterol 90 mcg MDI	Inhaled 4 times daily as needed for wheezing	6 mo prior	Has not refilled	Approximately once a month	Dr. Magaski
Epinephrine 0.3 mg/0.3 mL autoinjector	0.3 mL IM as needed for bee stings	1 yr prior	Has not refilled	Has not used	Dr. Magaski
Multivitamin	1 gummy po daily	02/2013		Yes	OTC per mother

IM = intramuscularly; MDI = metered dose inhaler; OTC = over the counter.

IMMUNIZATIONS:

HepB: Birth, 2, and 6 months

Rotavirus: 2, 4, and 6 months

DTaP: 2, 4, 6, and 15 months

Hib: 2, 4, and 6 months

IPV: 2, 4, and 6 months

PCV13: 2, 4, 6, and 15 months

MMR and varicella: 15 months

HepA: 15 and 24 months

Inactivated influenza: Received vaccine for this season

ALLERGIES: Bee stings (anaphylactic)

PHYSICAL EXAM (from clinic)

BP 100/60 mm Hg | Pulse 85 beats per min
Temp 98.3°F (oral) | RR 24 breaths per min
Wt 15.5 kg | Ht 98 cm | SpO_2 99%

GENERAL APPEARANCE: In mild-to-moderate pain (6/10 on visual analogue scale)

EYES: PERRLA, EOMs intact, conjunctiva clear

EARS: TM clear bilaterally

THROAT: Moist MM, no redness or swelling in throat

NECK: No lymphadenopathy

LUNGS: No crackles or wheezing

HEART: RRR, no gallops or murmurs

ABDOMEN: Tender to palpation, mild distension, no mass palpated

MUSCULOSKELETAL: Well perfused, no edema, no deformations

SKIN: No rashes present

NEUROLOGICAL: Alert and oriented

DRE: No masses or fecal impaction noted

LABORATORY DATA

None

DIAGNOSTIC TESTS

Stool culture (previous clinic visit): no growth, occult blood (-)

PROBLEM LIST

- ❑ Constipation
- ❑ Asthma
- ❑ Anaphylactic allergy

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SELF-ASSESSMENT QUESTIONS

1. What are the subjective and objective signs of constipation in this patient?
2. Classify this patient's constipation. What are the possible causes of constipation in this patient?
3. Why is this patient a candidate for OTC treatment for his constipation?
4. What nonpharmacologic treatment options are recommended for this patient?
5. What pharmacologic treatment options are recommended for this patient?
6. Devise a pertinent patient education plan for the caregiver.
7. Identify any medication-related problems.

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CASE 5.2
Crohn Disease | Level 2

Jeremy S. Stultz and Milap C. Nahata

LEARNING OBJECTIVES

1. Describe the pertinent signs and symptoms suggesting active Crohn disease (CD) in an adolescent patient.
2. Identify any medication-related problems and make appropriate interventions.
3. Synthesize appropriate pharmacologic and nonpharmacologic treatments of CD.
4. Develop appropriate monitoring parameters and treatment goals for CD.

CHIEF COMPLAINT: Bloody stools, diarrhea, and fatigue

History of Present Illness: This patient is a 16-year-old Caucasian female who was diagnosed with CD 1 year ago. She was most recently seen at the clinic 1 month ago and prescribed prednisone 30 mg bid for 2 weeks, followed by a taper over 4 weeks. She is currently in the middle of the fourth week of this treatment but came back to the gastrointestinal clinic today because her original symptoms have returned (i.e., abdominal pain, bloody diarrhea, and fatigue). Since her diagnosis, this is her second corticosteroid burst treatment needed to induce remission of her disease. She denies sexual activity, drug use, and hematuria. She was referred from the clinic to the hospital for further workup.

Review of Systems: Right lower quadrant abdominal pain, fatigue, and melena

Birth History: Unremarkable

Past Medical History: CD, depression (diagnosed 6 months PTA)

Past Surgical History: None

Social History: Lives with mother and younger brother, parents are divorced, currently in 11th grade (fall semester)

Family History: Father—HTN, hyperlipidemia; mother—healthy for age

Diet: Limited currently due to presenting symptoms

Immunizations:

Hepatitis B: Three doses before 18 months

DTaP: Five doses (last at 6 years)

Tdap: Age 12

Hib: Three doses before 18 months

IPV: Four doses (last at 6 years of age)

MMR: Two doses (last at 6 years)

Influenza trivalent vaccine: Annually, has not received one this year

Hepatitis A: Two doses before 2 years

Allergies: NKDA

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MEDICATION HISTORY

Medication	Sig	Start Date	End Date	Taking	Authorizing Provider
Mesalamine	1,000 mg po 4 times daily	6 mo PTA		Yes	Dr. Winthrop
Mesalamine	750 mg po 4 times daily	1 yr PTA	6 mo PTA	No	Dr. Winthrop
Prednisone	30 mg po bid x 14 days, 20 mg bid x 7 days, 10 mg bid x 7 days, 10 mg daily x 7 days, 5 mg daily x 7 days	4 wk PTA		Yes	Dr. Winthrop
Prednisone	30 mg po bid x 14 days, 20 mg bid x 7 days, 10 mg bid x 7 days, 10 mg daily x 7 days, 5 mg daily x 7 days	8 mo PTA	5.5 mo PTA	Yes	Dr. Winthrop
Ethinyl estradiol/ drospirenone	1 tab po daily	3 yr PTA		Yes	Dr. Ogana
Loperamide	2 mg with each loose stool	3 days PTA		Yes	Self-prescribed
Ibuprofen	400 mg 4 times daily prn pain	3 days PTA		Yes	Self-prescribed
Sertraline	100 mg po daily	6 mo PTA		Yes	Dr. Ogana

PTA = prior to admission.

PHYSICAL EXAM

BP 122/83 mm Hg | Pulse 75 beats per min
Temp 37.9°C (oral) | RR 18 breaths per min
Wt 41 kg | Ht 151 cm | SpO_2 99% on room air
LMP (2 weeks PTA)

General Appearance: Patient appears fatigued, mildly malnourished, and with apparent abdominal discomfort (pain score 8/10 on numerical rating scale)

Eyes: PERRLA, EOMs intact

Ears: TMs clear bilaterally

Throat: Clear mucous membranes

Neck: No lymphadenopathy

Lungs: CTA bilaterally, no retractions or nasal flaring

Heart: RRR, no murmurs/gallops/clips

Abdomen: Pain with palpation

Musculoskeletal: No edema, capillary refill 2 seconds

Skin: No rashes, petechiae, or purpura; present perianal fissures, and two 6-mm perianal skin tags

Neurological: Strength 5/5 upper and lower extremity, sensation and reflexes normal, interacting appropriately although fatigue noted

PCDAI score (based on reported symptoms and labs): 42 (moderate to severe)

LABORATORY DATA (on admission)

BASIC METABOLIC PANEL		
Component	**Value**	**Range**
Glucose	100	60–110 mg/dL
BUN	19	7–17 mg/dL
Sodium	137	137–145 mmol/L
Potassium	3.4	3.6–5.0 mmol/L
Chloride	96	98–107 mmol/L
CO_2	24	22–30 mmol/L
Anion gap	17	6–16 mmol/L
Creatinine	0.8	0.60–1.00 mg/dL
Calcium	7.4	8.4–10.2 mg/dL
Magnesium	1.7	1.6–2.3 mg/dL
Albumin	3.0	3.5–5.0 g/dL
CBC WITH DIFF		
WBC count	19.0	4.5–11.0 x 10^3/µL
RBC count	3.4	4.30–5.10 million/µL
Hemoglobin	10.2	11.4–15.4 g/dL

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LABORATORY DATA (cont'd)

CBC WITH DIFF (cont'd)		
Hematocrit	32.5	36.0% to 49.0%
MCV	76.2	78.0–98.0 fL
MCH	25.2	26.0–34.0 pg
MCHC	33.1	32.0–35.0 g/dL
RDW	13	11.5% to 14.5%
ESR	100	<15 mm/hr
CRP	1.2	<1.2 mg/dL
Platelets	550	150–450 x 10^3/µL
MPV	10.3	9.4–12.4 fL
Neutrophils	64.5	34.0% to 71.1%
Lymphocytes	22.5	19.3% to 51.7%
Monocytes	8.2	3.0% to 13.0%
Eosinophils	2.8	0.7% to 5.8%
Basophils	1.0	0.1% to 1.2%
PREGNANCY, URINE, STOOL		
Pregnancy test	(-)	
Fecal calprotectin	250	<50 mcg/g
Stool *C. diff* toxin	(-)	
Guaiac stool test	(+)	

BUN = blood urea nitrogen; CBC = complete blood count; CO_2 = carbon dioxide; CRP = C-reactive protein; ESR = erythrocyte sedimentation rate; MCH = mean corpuscular hemoglobin; MCHC = mean corpuscular hemoglobin concentration; MCV = mean corpuscular volume; MPV = mean platelet volume; RBC = red blood cell; RDW = red cell distribution width; WBC = white blood cell.

DIAGNOSTIC TESTS

Stool culture: NGTD

Colonoscopy with biopsy: Cobblestoning in the ileum and colon

UGI w/ SBFT: Linear ulceration with cobblestoning in the ileum and colon

MEDICATIONS AT ADMISSION

D10%W + 0.45% NaCl + 20 mEq/L KCl IV continuous infusion @ 100 mL/hr

Mesalamine: 1,000 mg (4 × 250 mg capsules) po bid

Prednisone: 10 mg tablet po bid

Ibuprofen: 400 mg tablet po q 4 hr prn for pain

Acetaminophen: 500 mg tablet po q 6 hr prn for fever

Loperamide: 2 mg tablet po with each loose stool

Sertraline: 100 mg tablet po daily

PROBLEM LIST

- ❑ Moderate-to-severe active CD
- ❑ Malnourishment
- ❑ Preventative medicine
- ❑ Depression

SELF-ASSESSMENT QUESTIONS

1. What subjective and objective evidence suggests active CD in this patient?
2. What risk factors does this patient have for the development of active CD?
3. Assess the current pharmacologic management of this patient's CD.
4. Develop a pharmacologic plan to induce remission for this patient's CD.
5. Offer potential interventions on any medication-related problems for this patient.
6. Include specific monitoring parameters for efficacy and safety of this patient's treatment.
7. Suggest nonpharmacologic treatment options.
8. Recommend appropriate immunizations for this patient.

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CASE 6.1
Nephrotic Syndrome | Level 2

Sandra Benavides

LEARNING OBJECTIVES

1. Identify common presenting signs and symptoms of nephrotic syndrome.
2. Recognize and develop therapeutic strategies for the complications of nephrotic syndrome.
3. Design a monitoring plan for efficacy and toxicity of pharmacologic therapy for nephrotic syndrome.
4. Determine appropriate outcomes for considering the discontinuation of pharmacologic therapy in nephrotic syndrome.
5. Identify important health maintenance education points for patients with nephrotic syndrome.

CHIEF COMPLAINT: Follow-up for kidneys

History of Present Illness: A 17½-year-old Hispanic male diagnosed with nephrotic syndrome at 5 years of age presented to nephrology clinic for follow-up. His initial years of diagnosis were characterized by frequently relapsing nephrotic syndrome. Approximately 3 years ago, he was treated with cyclosporine for a period of time due to frequent relapses. However, when he had a relapse while on cyclosporine, he developed severe uremia and neurologic symptoms of uremia within 3 months of treatment. Thereafter, the cyclosporine was discontinued and he was started on CellCept. On that, he was free of edema for a few years. However, he recently recovered from a major relapse characterized by anasarca, a very high hemoglobin (up to 19 g/dL), and hyponatremia. He was admitted to the local children's hospital, first rehydrated with saline and then eventually with 25% albumin. He was released but then readmitted shortly thereafter because of difficulty in controlling the relapse. He received multiple doses of Solu-Medrol infusions and completed a four-dose course of rituximab infusions. He went into a prolonged remission after that for approximately 2 years. However, he became nonadherent with his medications and was seen last month for relapse. At that time, he was found to be hemoconcentrated and was readmitted to the hospital for 5 days; he continued to have complications related to hyponatremia, decreased urine output, and marked anasarca. He received two doses of Solu-Medrol and one dose of albumin followed by Lasix, which he seemed to respond to, and then he underwent the rituximab treatment (two doses on this course). The patient, however, was released a week ago even though his anasarca remained because of psychosocial issues. He requires Lasix once a day and feels that the edema has somewhat improved but not subsided. His mother states that he started to have a cold during this time.

Review of Systems: Patient complains of swelling and mild abdominal discomfort but no respiratory distress. He reports a runny nose and headache but no sore throat, fever, or cough.

Past Medical History: Nephrotic syndrome (10 years); allergic rhinitis (4 years)

Past Surgical History: None

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MEDICATION HISTORY

Medication	Sig	Start Date	End Date	Taking	Authorizing Provider
Mycophenolate	250 mg po bid	2 yr prior	1 wk ago	No	Dr. Abbott
Mycophenolate	500 mg po bid	Dose increased at last admission	1 wk ago	Yes	Dr. Abbott
Vitamin D	1,000 units po daily	1 yr prior		Yes	Dr. Abbott
Montelukast	5 mg po hs	4 yr		Yes	Dr. Borerro
Prednisone	60 mg po daily	2 yr prior		Yes	Dr. Abbott
Enalapril	10 mg po daily	1 yr		Yes	Dr. Abbott
Simvastatin	10 mg po daily	1 yr		Yes	Dr. Abbott
Ibuprofen	400 mg po q 6 hr as needed for headache	1 wk		Yes	Self-administered
Furosemide	20 mg po daily	1 wk		Yes	Dr. Abbott

Social History: Lives with parents and attends school, denies alcohol and drug use

Family History: No known history of kidney disease

Immunizations: UTD, negative for seasonal influenza vaccine (refuses)

Allergies: NKDA

PHYSICAL EXAM

BP 138/94 mm Hg | Pulse 106 beats per min
Wt 77.8 kg (between 75th and 85th percentile)
Ht 179.5 cm (between 50th and 75th percentile)
BMI 24.1 kg/m^2 (75th percentile)

General Appearance: Alert, no facial swelling, but significant pitting edema on both legs and ascites

HEENT: Mild pharyngeal injection, some rhinorrhea, no lymphadenopathy; eyes normal; had an eye exam this year and it was WNL

Neck: The neck is supple, no lymphadenopathy, no thyromegaly

Heart: RRR, no murmur

Lungs: Lungs sound clear, no wheezes, no rales

Abdomen: Soft, positive for ascites but not tender

GU: No scrotal swelling

Musculoskeletal: Pitting edema 2+ up to his knees

Skin: Skin showed keloids over both ears from piercings

Neurological: Nonfocal

LABORATORY DATA

BASIC METABOLIC PANEL		
Component	**Value**	**Range**
Glucose	84	60–110 mg/dL
BUN	7	7–17 mg/dL
Sodium	143	137–145 mmol/L
Potassium	3.8	3.6–5.0 mmol/L
Chloride	106	98–107 mmol/L
CO_2	27	22–30 mmol/L
Creatinine	0.52	0.60–1.00 mg/dL
Calcium	9.6	8.4–10.2 mg/dL
Phosphorus	3.4	2.5–4.5 mg/dL
Albumin	2.1	3.5–5 g/dL
CBC		
WBC count	3.8	4.5–11.0 x 10^3/µL
RBC count	4.73	4.30–5.10 million/µL
Hemoglobin	14.3	11.4–15.4 g/dL
Hematocrit	43.1	36.0% to 49.0%
Platelets	220	150–450 x 10^3/µL

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LABORATORY DATA (cont'd)

CBC (cont'd)		
MCV	91	78–98 fL
MCH	30.2	26–34 pg
MCHC	33.2	32–35 g/dL
FASTING LIPID PROFILE		
Total cholesterol	136	125–170 mg/dL
Triglyceride	77	33–129 mg/dL
HDL	45	38–76 mg/dL
LDL	76	≤110 mg/dL
Urine		
Microalbumin, random	186	0–17 mcg/mL
Creatinine, random	274.1	24–392 mg/dL
Protein total, urine	38.7	0–15 mg/dL
Protein/creatinine ratio	0.14	<0.2
Other		
Vitamin D, 25-hydroxy	22.7	30–100 ng/mL

BUN = blood urea nitrogen; CBC = complete blood count; CO_2 = carbon dioxide; HDL = high-density lipoprotein; LDL = low-density lipoprotein; MCH = mean corpuscular hemoglobin; MCHC = mean corpuscular hemoglobin concentration; MCV = mean corpuscular volume; RBC = red blood cell; WBC = white blood cell.

RENAL BIOPSY

Positive for focal segmental glomerulosclerosis

PROBLEM LIST

- ❑ Nephrotic syndrome (steroid-dependent, frequently relapsing, in remission)
- ❑ Hypertension
- ❑ Vitamin D deficiency

SELF-ASSESSMENT QUESTIONS

1. What are the subjective and objective signs and symptoms of nephrotic syndrome in the patient?
2. Describe the treatment goals and outcomes in the patient.
3. Evaluate the appropriateness of each treatment strategy for nephrotic syndrome in the patient and recommend any necessary changes to his regimen.
4. Identify and resolve the medication-related problem(s).
5. Describe short-term and long-term adverse reactions to corticosteroids and management of adverse reactions.
6. List other patient counseling points important to communicate with the caregiver and patient.

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CASE 6.2
Acute Kidney Injury | Level 3

Jeremy S. Stultz and Milap C. Nahata

LEARNING OBJECTIVES

1. Identify risk factors for and evidence of acute kidney injury (AKI).
2. Categorize the severity of a patient's AKI.
3. Identify medication-related problems.
4. Synthesize medication-related plans to improve renal insufficiency and alleviate harm due to renal insufficiency.
5. Develop monitoring parameters for a patient with AKI.

CHIEF COMPLAINT: Worsening fever, SOB, and fatigue for 10 days and unresponsive on the day of admission (36 hours ago)

History of Present Illness: The patient is an 8-year-old female with a 10-day history of wheezing, SOB, increased work of breathing, fatigue, and a dry, hacking cough. On day 3, she was started on cefdinir for a community-acquired pneumonia. She felt better for a few days, but the fevers persisted and her mother recorded an oral temperature of 104°F the day before admittance (day 6 of cefdinir treatment). Her mother stated that the patient had a decreasing appetite over the past few days. Her symptoms continued to worsen; she started "talking funny" and eventually became unresponsive yesterday afternoon. The mother called 911 and the patient was brought directly to the hospital. On admission, she was described as being in warm septic shock and intubated. Blood cultures were drawn (before the first dose of antibiotics), antibiotics were started, and she was sent to the pediatric intensive care unit. Inotropic support has been needed to maintain hemodynamic stability. A chest x-ray revealed an overwhelming right-sided pneumonia.

Review of Systems: Intubated and sedated patient

Birth History: Full term, vaginal

Past Medical History: Mild persistent asthma (well controlled, last asthma admission 13 months ago, discharged from the ED), allergic rhinitis

Past Surgical History: None

Social History: Lives with parents (married 20 years; mother is 40 and father is 42) and two older siblings (brother is 11 and sister is 15)

Family History: Brother—asthma; mother and father—healthy

Diet: Normal for age, until current issue (see History of Present Illness)

Immunizations: Up to date per records on file, received influenza vaccine this year

Allergies: Environmental, shellfish (hives, swelling)

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MEDICATION HISTORY

Medication	Sig	Start Date	End Date	Taking	Authorizing Provider
Cetirizine (1 mg/mL)	2.5 mg (2.5 mL) po qhs	3 yr ago		Yes	Dr. Ferriat
Fluticasone 100 mcg/ Salmeterol 50 mcg	2 inhalations twice daily	08/10/2012		Yes	Dr. Wilmer
Albuterol	4 times daily prn wheezing	08/10/2012		Yes	Dr. Wilmer
Cefdinir (125 mg/5 mL)	175 mg (7 mL) po bid x 7 days	7 days PTA		Completed	Dr. Ferriat
Multivitamin	po daily in a.m.	3 yr ago		Yes	Dr. Ferriat

PTA = prior to admission.

PHYSICAL EXAM

On admission:

BP 75/35 mm Hg | Pulse 150 beats per min (bounding) | Temp 39.0°C (oral) | RR 13 breaths per min | Wt 25 kg | Ht 125 cm SpO_2 78% on room air | SvO_2 71% | capillary refill <1 second

Today:

BP 100/62 mm Hg | Pulse 100 beats per min Temp (max) 37.8°C (rectal) | RR 20 breaths per min (ventilated) | Wt 26.7 kg | Ht 125 cm SpO_2 95% (intubated) | $ScvO_2$ 85% | capillary refill 1 second

I/O (PAST 24 HOURS): 2,900 mL/350 mL

UOP (PAST 24 HOURS): 325 mL

UOP (PAST 12 HOURS): 100 mL

LDA: Intubated, double lumen PICC, arterial line, NG tube

GENERAL APPEARANCE: Patient is intubated and sedated

EYES: Pupils dilated, minimal reactivity to light

EARS: TMs clear bilaterally

THROAT: Clear mucous membranes, currently intubated

NECK: Moderate lymphadenopathy present

LUNGS: Intubated, rails still noted

HEART: RRR

ABDOMEN: Distended, bowel sounds faint but noted

MUSCULOSKELETAL: Edematous, warm, dry extremities

SKIN: Pitting edema noted, no pressure sores noted

NEUROLOGICAL: Adequately sedated, minimal pain/agitation reported per nursing

DIAGNOSTIC TESTS

BLOOD AND SPUTUM CULTURES ON ADMISSION: Gram + cocci in clusters in 2/2 bottles (preliminary)

CHEST X-RAY: Extensive pulmonary edema consistent with RLL and RUL pneumonia

ECG: No abnormal rhythms, QTc 390 ms

MEDICATIONS SINCE ADMISSION

COMPLETED MEDICATIONS

Rocuronium (10 mg/mL) 25 mg IV once

Ketamine (10 mg/mL) 25 mg IV once

Lidocaine (10 mg/mL) 25 mg IV once

0.9% NaCl 500 mL bolus × 3 on admission

0.9% NaCl at 100 mL/hr (D/C'd after 3 hours)

Gentamicin (10 mg/mL) 62 mg IV once

Meropenem (20 mg/mL in D5%W) 1,000 mg IV once

Vancomycin (5 mg/mL in D5%W premixed) 375 mg IV once over 1 hour

Cosyntropin (0.25 mg/mL) 0.25 mg IV once

Midazolam IV (5 mg/mL) IV continuous infusion, max rate of 0.8 mL/hr = 0.16 mg/kg/hr

Fentanyl IV (10 mcg/mL) IV continuous infusion, max rate of 10.0 mL/hr = 4 mcg/kg/hr

Norepinephrine (16 mcg/mL) IV continuous infusion, max rate of 37.5 mL/hr = 0.4 mcg/kg/min

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LABORATORY DATA

BASIC METABOLIC PANEL				
Component	**Admission (6 mo ago)**	**Admission**	**Today**	**Range**
Glucose	95	150	220	60–110 mg/dL
BUN	17	32	33	8–19 mg/dL
Sodium	142	137	133	138–145 mmol/L
Potassium	4.2	3.5	5.4	3.4–5.2 mmol/L
Chloride	98	99	97	98–107 mmol/L
CO_2	18	19	17	20–28 mmol/L
Anion gap		19	21	6–16 mmol/L
Creatinine, serum (IDMS traceable)	0.42	0.51	1.2	0.20–0.70 mg/dL
Calcium	9.2	8.6	8.2	8.9–10.1 mg/dL
Phosphorus	5.3	5.4	6	3.8–5.5 mg/dL
Albumin	3.9	3.3	3.2	3.5–5 g/dL
iCa, arterial		1.4	1.1	1.2–1.35 mmol/L
pH, arterial		7.32	7.29	7.35–7.45
pCO_2, arterial		38	36	33–43 mm Hg
pO_2, arterial		80	80	80–105 mm Hg
TCO_2, arterial		17	16	18–29 mmol/L
Cortisol, plasma		15-pre, 21-post		>18–20 mcg/dL post
Lactate		2.5	2.4	0.5–2.2 mmol/L
CRP		3.5	2.5	<1.2 mg/dL
CBC WITH DIFF				
WBC count	8.6	19.8	18.8	4.5–11.0 x 10^3/µL
RBC count	4.8	4.4	4.3	4.30–5.10 million/µL
Hemoglobin	13.1	12	11.5	11.4–15.4 g/dL
Hematocrit	41.3	38.8	37.2	36.0% to 49.0%
MCV	85.2	82.0	81.2	78.0–98.0 fL
MCH	29.0	28.0	26.5	26.0–34.0 pg
MCHC	33.2	34.0	33.1	32.0–35.0 g/dL
RDW	12.3	12.0	11.6	11.5% to 14.5%
MPV	10.5	10.2	10.4	9.4–12.4 fL
Platelets	240	460	470	150–450 x 10^3/µL
Neutrophils		73.8	75.3	34.0% to 71.1%
Bands		14.9	13.2	5.0% to 11.0%
Lymphocytes		7.2	7.7	19.3% to 51.7%
Monocytes		3.1	2.8	3.0% to 13.0%
Eosinophils		0.8	0.7	0.7% to 5.8%
Basophils		0.2	0.3	0.1% to 1.2%
Urinalysis				
Na			52	<40 mEq/L
Creatinine, urine			22 mg/dL	
Appearance			Brown, granular casts present	
WBC			3+	Negative
RBC			2+	Negative
Protein			+	Negative
Glucose			1+	Negative

BUN = blood urea nitrogen; CBC = complete blood count; CO_2 = carbon dioxide; MCH = mean corpuscular hemoglobin; CRP = C-reactive protein; iCa = ionized calcium; IDMS = isotope dilution mass spectrometry; MCHC = mean corpuscular hemoglobin concentration; MCV = mean corpuscular volume; MPV = mean platelet volume; Na = sodium; pO_2 = partial pressure of oxygen; pCO_2 = partial pressure of carbon dioxide; RBC = red blood cell; RDW = red cell distribution width; TCO_2 = total carbon dioxide; WBC = white blood cell.

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Active medications

D5%W + 0.9% NaCl + 20 mEq/L KCl IV continuous infusion at 67 mL/hr

Acetaminophen (160 mg/5 mL) 352 mg NG q 6 hr prn fever >38.0°C

Albuterol 0.083% (2.5 mg/mL nebulization solution) ET tube q 3 hr

Albuterol 0.083% (2.5 mg/mL nebulization solution) ET tube q 3 hr prn wheezing

Calcium chloride (100 mg/mL) 500 mg IV q 3 hr prn via central line (for iCa <1.2 mg/dL)

Midazolam IV (5 mg/mL) IV continuous infusion at 0.5 mL/hr = 0.1 mg/kg/hr

Midazolam IV (5 mg/mL) 0.5 mL = 0.1 mg/kg/dose, q 1 hr prn agitation (two doses in the past 24 hours)

Fentanyl IV (10 mcg/mL in NS) continuous infusion at 5 mL/hr = 2 mcg/kg/hr

Fentanyl IV (10 mcg/mL in NS) 5 mL = 2 mcg/kg/dose q 1 hr prn pain (three doses in the past 24 hours)

Gentamicin (10 mg/mL) 62 mg IV q 8 hr

Meropenem (20 mg/mL in D5%W) 1,000 mg IV q 8 hr

Norepinephrine (16 mcg/mL in D5%W) IV continuous infusion at 9.4 mL/hr = 0.1 mcg/kg/min

PediaSure® NG continuous at 10 mL/hr

Ranitidine (25 mg/mL) 30 mg IV q 8 hr

Vancomycin (5 mg/mL in D5%W premixed) 375 mg IV q 6 hr, given over 1 hour

PROBLEM LIST

- ❑ AKI (likely ATN)
- ❑ Pneumonia with septicemia (improving septic shock)
- ❑ Respiratory failure requiring intubation
- ❑ Sedation/analgesia

SELF-ASSESSMENT QUESTIONS

1. Identify any potential causes of AKI in this patient.
2. Identify the subjective and objective evidence suggesting AKI in this patient.
3. Assess AKI based on creatinine clearance, the pRIFLE criteria, and UOP.
4. Identify the management goals for this patient's AKI.
5. Create a pharmacotherapeutic plan for managing this patient while she has AKI. Offer potential interventions on any medication-related problems to ensure safe and effective treatment of this patient while she has AKI.
6. Devise appropriate monitoring parameters for management of this patient.

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CASE 7.1
Type 2 Diabetes Mellitus | Level 1

Sandra Benavides

LEARNING OBJECTIVES

1. Identify risk factors and presenting signs and symptoms for type 2 diabetes mellitus (T2DM) in children and adolescents.
2. Develop a treatment regimen for a patient newly diagnosed with T2DM.
3. Determine risk factors and treatment strategies for premature cardiovascular disease in a pediatric patient diagnosed with T2DM.
4. Develop a treatment plan for an obese patient.
5. Identify a medication-related problem and develop a plan for resolution of the problem.

CHIEF COMPLAINT: Referral from PCP to endocrinology clinic for obesity

History of Present Illness: The patient is a 12-year-old Hispanic male who presented with his parents to the endocrinology clinic for an evaluation of obesity. His mother reported her son had a 25-pound weight gain over the past 6 months. His most recent laboratory analysis showed an increased insulin level per the pediatrician. The patient has always been "big" for his age according to his mother. At 8 years of age, he was about 200 pounds.

Review of Systems: The patient denied headaches, dizziness, visual changes, abdominal pain, nausea, vomiting, constipation, diarrhea, polyuria, polydipsia, or nocturia. His mother reported he is always tired despite sleeping through the night with the bi-pap machine. She also stated this has been an issue since he started risperidone.

Birth History: Per his mother, she had "sugar problems" during the pregnancy, and the patient was large at birth.

Past Medical History: Sleep apnea; developmental delay; asthma; morbid obesity; bipolar affective disorder; atopic dermatitis; allergic rhinitis

Past Surgical History: Tonsillectomy and adenoidectomy (mother unsure of year)

Development History: Patient has a history of developmental delay per testing done prior to age of 3 years, at which time he was placed in a developmental pre-kindergarten school and received speech therapy.

Social History: He lives with his mother, stepfather, and four sisters ages 15, 9, 7, and 2 years. He is in sixth grade at a local public school and is enrolled in a special education class. He receives speech therapy and behavioral counseling in the aftercare program. Neither his mother nor stepfather work; they receive Supplemental Security Income (SSI) and live in government housing.

Family History: Maternal side of family has many members affected with allergies, diabetes, heart disease, hypertension, kidney disease (aunt), sickle cell, cancer, obesity, stroke, and mental health issues (MGM). Father is obese. His mother has asthma and depression. The youngest sibling has eczema.

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MEDICATION HISTORY

Medication	Sig	Start Date	End Date	Taking	Authorizing Provider
Fluticasone/salmeterol 100 mcg/50 mcg	1 inhalation bid	About 2 yr ago		Yes	Dr. Talavera
Montelukast 5 mg	1 tab po q hs	About 2 yr ago		Yes	Dr. Talavera
Mometasone nasal spray	1 puff in each nostril bid	About 2 yr ago		Yes	Dr. Talavera
Albuterol	2 puffs inhaled q 4 hr prn wheezing	About 2 yr ago		Yes	Dr. Talavera
Risperidone 4 mg	1 tablet po bid	6 mo ago		Yes	Dr. Henderson

Diet: (Obtained by nutritionist in clinic.) For breakfast, patient typically has cereal with whole milk, two eggs, and sausage. Some days he does not eat a snack between breakfast and lunch. For lunch, he has a bologna and cheese sandwich on white bread. For an afternoon snack, he has chips or a candy bar. For dinner, he likes pizza, chicken nuggets, and macaroni and cheese. He does not like vegetables or fruit. He rarely drinks water and has regular soda throughout the day. Stepdad states he sneaks food as well because they find food wrappers in his room.

Immunizations: UTD except for the influenza vaccine this year

Allergies: NKDA (skin test positive for shrimp)

PHYSICAL EXAM

BP 127/62 mm Hg | Pulse 84 beats per min RR 18 breaths per min | Wt 126 kg (>99th percentile) | Ht 165.5 cm (95th percentile)

General Appearance: Awake, alert, looks fatigued, obese

Heent: Normocephalic, PERRLA, EOM(I), moist oral mucosa

Lungs: CTA bilaterally

Heart: RRR, S1, S2 normal; no murmur auscultated

Abdomen: Soft, nontender, nondistended, no palpable masses; no HSM

Extremities: No clubbing, cyanosis, edema, free ROM × 4

Skin: No rashes, lesions, acanthosis nigricans noted around neck

LABORATORY DATA (FASTING)

BASIC METABOLIC PANEL		
Component	**Value**	**Range**
Glucose	185	60–110 mg/dL
BUN	13	7–17 mg/dL
Sodium	138	137–145 mmol/L
Potassium	3.9	3.6–5.0 mmol/L
Chloride	105	98–107 mmol/L
CO_2	28	22–30 mmol/L
Creatinine	0.8	0.60–1.00 mg/dL
AST	17	15–50 units/L
ALT	17	10–50 units/L
Total cholesterol	167	125–170 mg/dL
TG	70	33–129 mg/dL
LDL	121	≤110 mg/dL
HDL	40	38–76 mg/dL
Insulin	52	<23 micro-international units/mL
C-peptide	3.3	0.80–3.10 ng/mL
Hemoglobin A_{1c}	8.2%	<5.7%

ALT = alanine transaminase; AST = aspartate aminotransferase; BUN = blood urea nitrogen; CO_2 = carbon dioxide; HDL = high-density lipoprotein; LDL = low-density lipoprotein; TG = triglyceride.

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PROBLEM LIST

- ❑ T2DM
- ❑ Dyslipidemia
- ❑ Prehypertension
- ❑ Morbid obesity

SELF-ASSESSMENT QUESTIONS

1. What is the subjective and objective evidence for the diagnosis of T2DM?
2. Assess the need for therapy for dyslipidemia and prehypertension for this patient.
3. Develop a pharmacologic regimen for the treatment of T2DM.
4. Determine an appropriate treatment strategy for morbid obesity in this patient.
5. Identify the medication-related problem and develop a solution.
6. Devise the most pertinent patient education for the patient and caregiver.

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CASE 7.2
Hypothyroidism | Level 2

Shirin Madzhidova and Sandra Benavides

LEARNING OBJECTIVES

1. Identify presenting signs and symptoms of secondary hypothyroidism and the most probable etiology of the disease.
2. Recommend appropriate pharmacologic therapy for hypothyroidism.
3. Evaluate the treatment and management modalities for growth hormone (GH) deficiency.
4. Recognize medication- and disease-related complications requiring emergency intervention in the case.
5. Devise treatment goals and outcomes for each of the identified healthcare-related problems presented in the patient case.

CHIEF COMPLAINT: Increased fatigue, delayed growth

History of Present Illness: A 5-year-old male, with no significant past medical history, presented to the pediatrician's office with symptoms of gradually increased fatigue, weight gain, and constipation over the past 6 months. The patient's mother has also noticed he is growing at a very slow rate, looking considerably smaller than his peers. She denied any signs of malnutrition or serious illness in the past year. Laboratory screening performed at birth did not show any significantly abnormal values. The patient was seen by his primary care pediatrician for well-check visits but was lost to follow-up shortly after the age of 2 years.

Review of Systems: Diet and appetite varies; recent difficulty with movement, gait, and balance present; recent history of cold intolerance

Birth history: Mother nulliparous prior to conception; received proper prenatal care throughout pregnancy; all maternal screening tests negative, including GBS; patient delivered at 39 weeks 2 days; spontaneous vaginal delivery uneventful; mother did not smoke, drink, or use recreational drugs during pregnancy; newborn screen was negative

Past Medical History: Nonsignificant

Past Surgical History: None

Development History: No heel prick test performed at birth; last well-child check was at 24 months (mother was told that patient was appropriately meeting developmental milestones); sat unaided by 8 months and could walk at 19 months; speaking at least two words by 12 months; brushing his teeth and clothing himself by 3 years

Social History: The patient lives with his mother and two younger siblings. His mother works part-time outside of the home. He attends school (kindergarten) 5 days per week. His maternal grandmother occasionally watches him.

Family History: Mother—seasonal allergies; maternal grandmother—type 2 diabetes mellitus

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Diet: Does not follow a steady diet, picky eater, refuses vegetables, and rarely eats fruits; mother gives him whatever he agrees to eat, often junk food

Immunizations: Up to date per the mother including this season's influenza vaccine (received at the local public health department); electronic state registry confirms immunizations through 24 months

Allergies: NKDA

MEDICATION HISTORY: None

PHYSICAL EXAM

BP 85/52 mm Hg | Pulse 120 beats per min
Temp 98.5°F (oral) | RR 32 breaths per min
Wt 16.4 kg | Ht 98 cm | BMI 17.1 kg/m^2
SpO_2 98% (room air)

General Appearance: Apparent growth delay and gait disturbance

Eyes: PERRLA; fundoscopic exam negative for papilledema

Ears: No discharge or drainage noted in the external canals

Throat: Oral mucosa dry but without lesions; gums healthy; oropharynx without exudate

Neck: No palpable lymphadenopathy; small and soft thyroid gland on palpation

Lungs: CTABL; no wheezes, rhonchi, or crackles; no accessory muscle use

Heart: RRR, no murmurs or gallops

Abdomen: Bowel sounds are present; nontender, nondistended; spleen and liver are nonpalpable

Musculoskeletal: No edema or digital clubbing; muscle weakness noted on horizontal and vertical suspension; tightness of the hip adductors and knee extensors noted

Skin: No visible lesions or rashes; no jaundice

Neurological: Normocephalic, atraumatic

LABORATORY DATA

BASIC METABOLIC PANEL		
Component	**Value**	**Range**
Glucose	62	60–110 mg/dL
BUN	7	7–17 mg/dL
Sodium	139	137–145 mmol/L
Potassium	4	3.6–5.0 mmol/L
Chloride	102	98–107 mmol/L
CO_2	29	22–30 mmol/L
Anion gap	6	6–16 mmol/L
Creatinine	0.6	0.60–1.00 mg/dL
Calcium	8.7	8.4–10.2 mg/dL

Thyroid Function	Gonadotropins	GH Testing
TSH: 2.36 (0.34–4.82 micro-international units/mL)	FSH: 1.0 (1.4–18.1 milli-international units/mL)	Insulin-like GFB protein 3: 1.8 (2.7–8.9 mg/L)
T_4: 5.4 (4.5–12.1 mcg/dL)	LH: 1.0 (0.1–6.0 milli-international units/mL)	IGF-1 ECL: 134 (140–507 ng/mL)
Free T_4: 0.67 (0.8–2.0 ng/dL)		

BUN = blood urea nitrogen; CO_2 = carbon dioxide; ECL = electrochemiluminescent immunoassay; FSH = follicle-stimulating hormone; GFB = growth factor-binding; GH = growth hormone; IGF-1 = insulin-like growth factor; LH = luteinizing hormone; T_4 = thyroxine; TSH = thyroid-stimulating hormone.

DIAGNOSTIC TESTS

MRI of the sella/suprasellar areas: No visible masses or lesions are present.

Left hand and wrist radiography for bone age: Patient's skeletal age is felt to be approximately 3 years of age as compared with standards, which is inconsistent with the patient's stated chronological age.

PROBLEM LIST

- ❑ Secondary hypothyroidism
- ❑ Panhypopituitarism
- ❑ Growth deficiency
- ❑ Hypotonia

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SELF-ASSESSMENT QUESTIONS

1. Using Figures 1 through 3, what are the patient's current weight, height, and BMI percentiles?
2. What subjective and objective evidence supports the diagnosis of secondary hypothyroidism?
3. Devise a pharmacologic regimen for the treatment of hypothyroidism that is best suited for this patient.
4. Assess the pharmacologic therapy for the patient's GH deficiency and appropriate monitoring for clinical and biochemical responses to treatment.
5. Devise the most pertinent patient education for the caregiver regarding levothyroxine and GH administration, monitoring, and follow-up.
6. Five days post-initiation of therapy for patient's hypothyroidism, he presents to the ED with symptoms suggestive of hypoglycemia, dehydration, and altered mental status. Upon closer evaluation, the patient was found to be in adrenal crisis. Evaluate the potential drug or disease interactions that may have precipitated the adrenal crisis and the appropriate management strategies.
7. Identify a potential medication-related adverse reaction associated with GH replacement therapy and its management.

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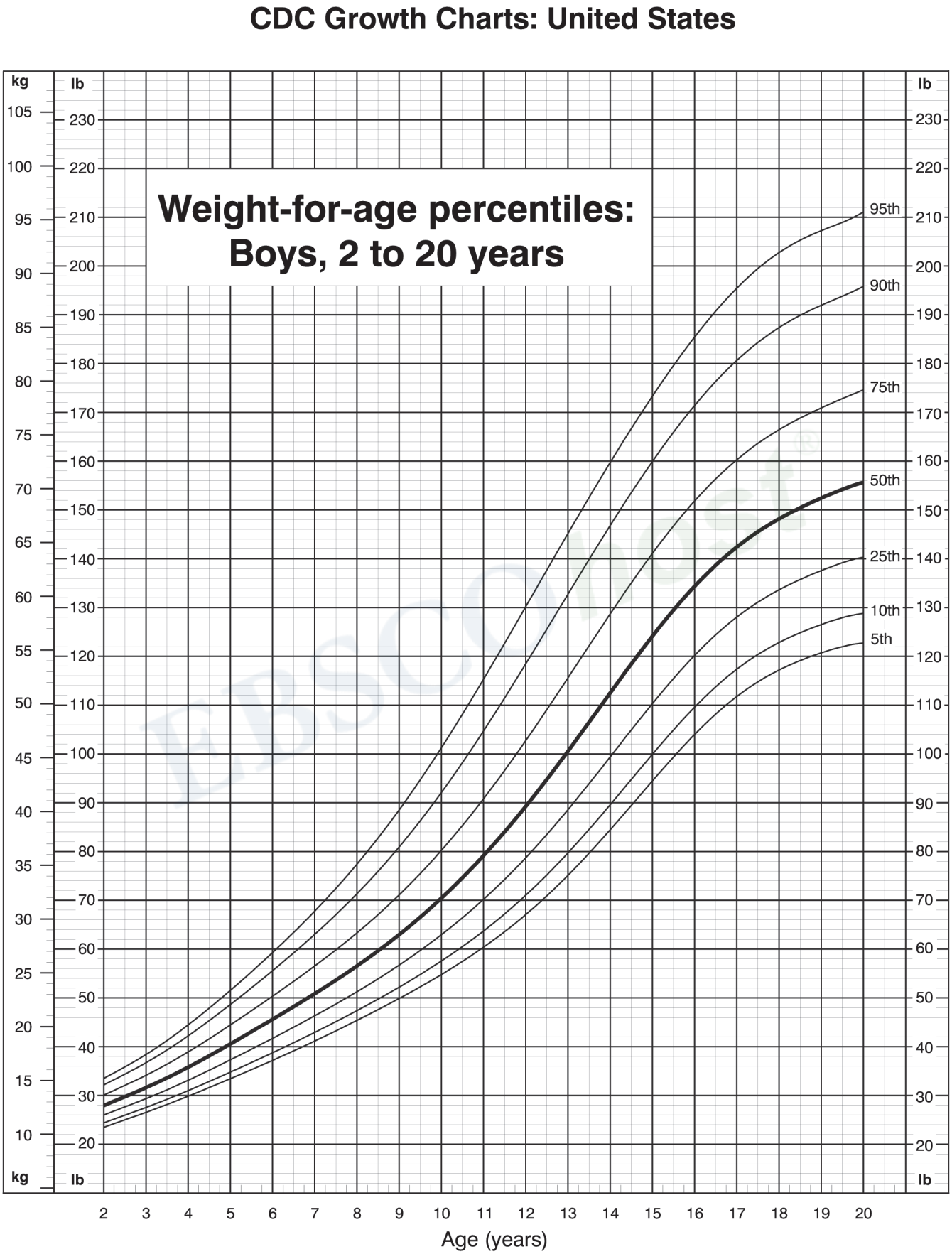

FIGURE 1: Weight-for-age percentiles, boys 2–20 years. *Source:* Kuczmarski RJ, Ogden CL, Guo SS, et al. 2000 CDC Growth Charts for the United States: methods and development. National Center for Health Statistics. *Vital Health Stat* 11. 2002;(246):1–190.

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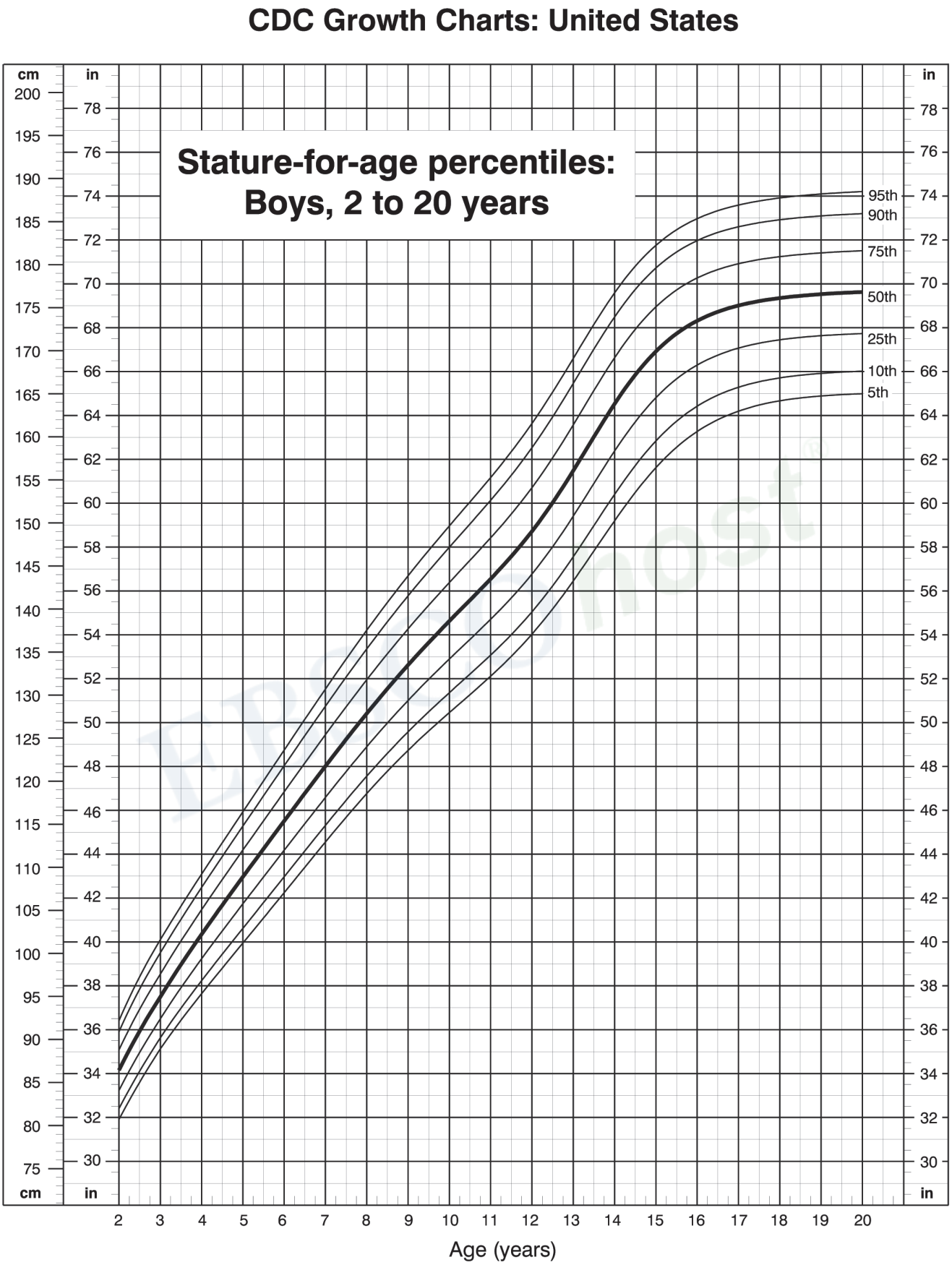

FIGURE 2: Stature-for-age percentiles, boys 2–20 years. *Source:* Kuczmarski RJ, Ogden CL, Guo SS, et al. 2000 CDC Growth Charts for the United States: methods and development. National Center for Health Statistics. *Vital Health Stat 11.* 2002;(246):1–190.

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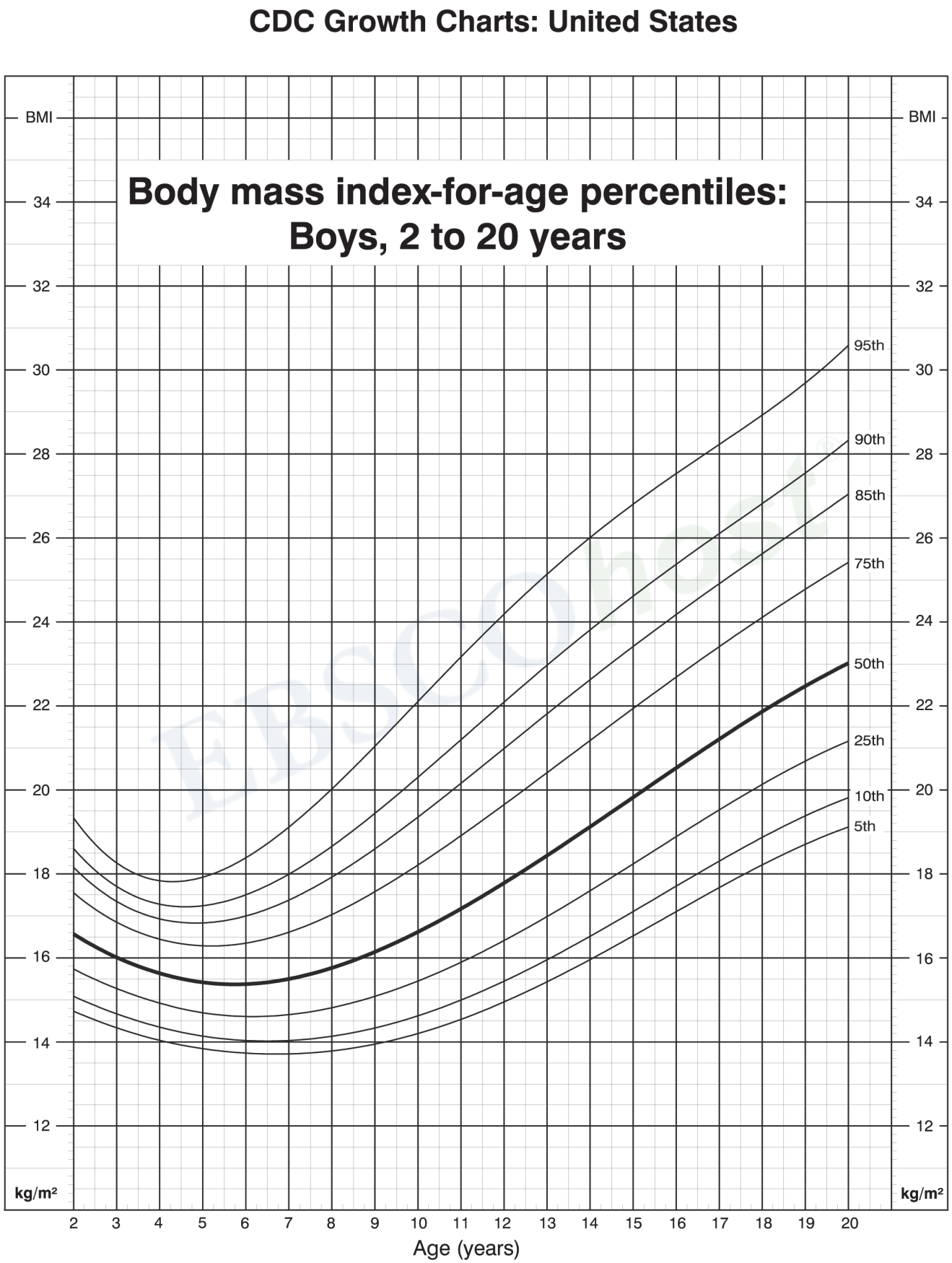

FIGURE 3: Body mass index-for-age percentiles, boys 2–20 years. *Source:* Kuczmarski RJ, Ogden CL, Guo SS, et al. 2000 CDC Growth Charts for the United States: methods and development. National Center for Health Statistics. *Vital Health Stat 11.* 2002;(246):1–190.

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CASE 7.3
Diabetic Ketoacidosis | Level 3

Julianna Crain and Sandra Benavides

LEARNING OBJECTIVES

1. Identify the presenting signs and symptoms of diabetic ketoacidosis (DKA) in a pediatric patient.
2. Identify diagnostic criteria and goals of therapy for DKA.
3. Assess the appropriate pharmacologic therapy for prevention and resolution of DKA.
4. Describe and recommend therapeutic endpoints and outcomes for each of the identified DKA-associated problems in the patient case.
5. Delineate necessary monitoring of therapy.

CHIEF COMPLAINT: Intractable vomiting

History of Present Illness: A 14-year-old female with a past medical history significant for type 1 diabetes mellitus arrived to the emergency department (ED) with an acute episode of hyperglycemia. Her home glucometer indicated an elevated serum glucose concentration of 420 g/dL on her home glucometer, and she tested positive for ketones in her urine. Two days prior to this admission, the patient presented to the ED with mild DKA. She received intravenous fluids (IVFs) and insulin, her symptoms resolved, and she was discharged home. At that time, her mother stopped her insulin pump because the patient has a history of severe hypoglycemia. The patient had nonstop emesis and chills since 6:30 p.m. the night prior to this current admission that prompted her to go to the ED for further evaluation and management. Other symptoms included abdominal pain, chest pain, headaches, and constipation. Her last bowel movement was 2 days prior to admission. Upon arrival, she was immediately transferred to the PICU for management of DKA.

Review of Systems: Positive for chills, vomiting, abdominal pain, chest pain, headaches, and constipation

Past Medical History: Type 1 diabetes mellitus diagnosed 2 years ago with six hospitalizations due to DKA; depression; POTS; gastroparesis

Developmental History: In the ninth grade, no history of developmental delay

Past Surgical History: Tonsillectomy and adenoidectomy 6 years ago; rods placed in left arm due to fracture last year after falling off a trampoline

Social History: Lives with mother, stepfather, biological sister, and two dogs; in the summer, she lives with her father, father's girlfriend, sister, and one dog in a different state; negative for tobacco exposure, alcohol, and drug use

Diet: Regular for age with limited concentrated sweets

Family History: Mother has POTS disease

Immunizations: Up to date, interested in flu shot prior to discharge

Allergies: NKDA

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HOME MEDICATION HISTORY

Medication	Sig	Start Date	End Date	Taking	Authorizing Provider
Regular insulin	Rate as instructed for insulin pump	11/2011		No	Dr. Steinberg
Glucagon emergency kit	1 mg SC prn unconsciousness or seizure from hypoglycemia	11/2011		Yes	Dr. Steinberg
Ondansetron	4 mg po q 8 hr prn vomiting	2 days		Yes	Dr. Perez
Fludrocortisone	0.05 mg po once daily	05/2007		Yes	Dr. Patel
Escitalopram	5 mg po once daily	03/2012		Yes	Dr. Velez
Cyproheptadine	4 mg po once daily	Cannot remember		Yes	Dr. Adams

SC = subcutaneous.

PHYSICAL EXAM

BP 83/49 mm Hg | Pulse 121 beats per min
RR 40 breaths per min | Wt 34.9 kg
Ht 175 cm | BMI 15.6 kg/m^2 | GCS score = 15

General Appearance: Awake, agitated, shaking

Head: Normocephalic, atraumatic

Eyes: PERRLA, EOMI, normal conjunctiva and lids, no discharge, slightly sunken

Ears, Nose, Mouth, and Throat: Dry mucous membranes and lips without lesions; bilateral ear canals and tympanic membranes normal, pharynx without redness, exudates, or edema

Neck: No meningismus, no masses

Cardiovascular: RRR, no murmur, no gallops, no rubs

Respiratory: Lungs CTA, Kussmaul breathing

Gastrointestinal: Decreased bowel sounds, abdominal distension and tenderness without organomegaly

Musculoskeletal: No deformity or swelling of the joints, moves all extremities without deficit, cool extremities

Skin: No lesions, rash, petechiae, or bruising noted, no jaundice, skin recoil 2 seconds

Neurological: Alert, no focal deficit observed, cranial nerves grossly intact

LABORATORY DATA

BASIC METABOLIC PANEL		
Component	**Value**	**Range**
Glucose	647	60–110 mg/dL
BUN	39	7–17 mg/dL
Sodium	130	137–145 mmol/L
Potassium	5.7	3.6–5.0 mmol/L
Chloride	96	98–107 mmol/L
CO_2	9.3	22–30 mmol/L
Anion gap	37	6–16 mmol/L
Creatinine	2.3	0.60–1.00 mg/dL
Calcium	10.9	8.4–10.2 mg/dL
Phosphorus	5.8	2.9–5.3 mg/dL
Magnesium	2.0	1.6–2.3 mg/dL
CALCULATED OSMOLALITY		
Osmolality	303	263–273 mOsm/kg
CBC WITH DIFF		
WBC count	7.02	4.5–11.0 × 10^3/μL
RBC count	4.93	4.30–5.10 million/μL
Hemoglobin	12	11.4–15.4 g/dL
Hematocrit	37.9	36.0% to 49.0%
MCV	93.2	78.0–98.0 fL
MCH	32	26.0–34.0 pg
MCHC	34.4	32.0–35.0 g/dL
RDW	12.3	11.5% to 14.5%
Platelets	343	150–450 × 10^3/μL
MPV	7.37	9.4–12.4 fL
Neutrophils	78	34.0% to 71.1%
Immature granulocytes	0	0.0% to 0.5%
Neutrophils absolute	5.5	1.56–6.13 × 10^3/μL
Lymphocytes	16	19.3% to 51.7%

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LABORATORY DATA (cont'd)

CBC WITH DIFF (cont'd)		
Lymphocytes absolute	1.1	1.18–3.74 × 10^3/μL
Monocytes	5	3.0% to 13.0%
Monocytes absolute	0.36	0.24–0.86 × 10^3/μL
Eosinophils	0	0.7% to 5.8%
Eosinophils absolute	0.02	0.04–0.36 × 10^3/μL
Basophils	0	0.1% to 1.2%
Basophils absolute	0.02	0.01–0.08 × 10^3/μL

URINALYSIS	
Component	**Value**
Urine total volume	60 mL
Color	Yellow
Turbidity	Clear
Specific gravity	1.025
pH	6
Nitrites	Negative
Leukocyte esterase	Negative
Urine protein	Negative
Urine glucose	500 mg/dL
Urine ketones	>80 mg/dL
Urobilinogen	0.2 mg/dL
Occult blood	Negative
WBC	None seen
RBC	0–2
Epithelial cells	0–8
Bacteria	Rare
Crystals	None seen
Casts	None seen
Mucus	None seen
Pregnancy test	Negative

VENOUS BLOOD GAS	
Component	**Value**
pH	7.14
pCO_2	25 mm Hg
HCO_3	9.8 mmol/L
BE	–18.8 mmol/L
SpO_2	86.1%

BE = base excess; BUN = blood urea nitrogen; CBC = complete blood count; CO_2 = carbon dioxide; HCO_3 = bicarbonate; MCH = mean corpuscular hemoglobin; MCHC = mean corpuscular hemoglobin concentration; MCV = mean corpuscular volume; MPV = mean platelet volume; pCO_2 = partial pressure of carbon dioxide; RBC = red blood cell; RDW = red cell distribution width; SpO_2 = oxygen saturation via pulse oximetry; WBC = white blood cell.

MEDICATIONS AT ADMISSION

NS 700 mL IV once, stat

NS 350 mL IV once, stat

Famotidine 16 mg IV q 12 hr for two doses

Fluzone quadrivalent vaccine 0.5 mL IM once

Ondansetron 4 mg IV once

Acetaminophen 480 mg IV q 4 hr prn mild pain, temp greater than or equal to 38.5˚C

Ketorolac 20 mg IV q 6 hr prn moderate pain (maximum 5 days)

0.45% NS + 40 mEq KCL at 115 mL/hr IV (day one) followed by

D5W + 0.45% NS + 40 mEq KCl at 115 mL/hr IV (day one) followed by

D10W + 0.45% NS + 40 mEq KCl at 115 mL/hr IV (day one) and

Insulin regular 50 units + 0.9% saline at 3.5 mL/hr IV (day one)

PROBLEM LIST

- ❑ DKA
- ❑ Dehydration

SELF-ASSESSMENT QUESTIONS

1. What subjective and objective evidence supports the diagnosis of DKA in this patient?
2. Describe the necessary laboratory parameters to obtain before developing an appropriate pharmacologic regimen for this patient.
3. What are the overall goals of therapy related to DKA? Given these goals, develop a pharmacologic regimen for the treatment of DKA.
4. Describe appropriate management of type 1 diabetes mellitus during an illness and potential strategies to avoid DKA in the future.
5. Describe the importance of immunizations in this patient.
6. Identify and resolve the medication-related problem(s).

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CASE 8.1
Attention Deficit Hyperactivity Disorder | Level 1

Jennifer E. Thomas and Joshua Caballero

LEARNING OBJECTIVES

1. Identify the signs and symptoms of attention deficit/hyperactivity disorder (ADHD) and oppositional defiant disorder (ODD) in a pediatric patient.
2. Identify environmental risk factors associated with ADHD.
3. Recommend an appropriate treatment regimen for the patient presented in this case.
4. Assess patient response to pharmacologic therapy and identify any medication-related problems that require adjustment of therapy.
5. Identify counseling points on pharmacologic therapy that should be discussed with the patient or caregiver.

CHIEF COMPLAINT: Increase in aggressive behaviors and insomnia

History of Present Illness: An 11-year-old white male presented to the pediatrician after complaints from his school regarding his disruptive behavior. His teacher reported frequent anger outbursts and that he starts fights with other students. He is able to concentrate on an assignment only for a few minutes at a time and frequently makes careless errors. His parents state he often does not seem to listen when they speak to him and that he has a hard time following directions and completing homework without becoming distracted. He has also had an increase in bouts of anger at home in which he throws things and hits his younger sister. During his recent birthday, he became angry at his sister for blowing out his candles and struck her with his fist. The patient was diagnosed with ADHD at the age of 7, which had been controlled with amphetamine/dextroamphetamine XR for 2 years. The dose was increased last month from 25 mg to 30 mg daily after the patient experienced a decline in ability to concentrate compared to the past 2 years. There has not been any improvement in ADHD symptoms, yet there has been a rise in difficulty sleeping since the dose was increased. Aggression has also not improved.

Birth History: Delivery via C-section at full term; no complications; birth weight of 5 lb 4 oz and birth length of 50 cm

Past Medical History: ADHD, ODD; psychological behavioral therapy failed

Past Surgical History: None

Developmental History: All milestones appropriately met

Social History: Denies alcohol, smoking, or illicit drug use

Family History: Mother—postpartum depression following both pregnancies. Father—none; however, wife describes him as having difficulty completing projects around the house (e.g., unfinished basement, unfinished tree house). Sister—7 years old, no medical issues noted.

Diet: Eats lunch served at the school cafeteria; eats dinner mostly at home; family meals include fried foods and processed products (e.g., boxed mac-and-cheese, instant mashed potatoes).

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MEDICATION HISTORY

Medication	Sig	Start Date	End Date	Taking	Authorizing Provider
Amphetamine/dextroamphetamine XR 30 mg	Take once daily	08/01/13		Yes	Dr. Winchester
Amphetamine/dextroamphetamine XR 25 mg	Take once daily	03/18/11	07/31/13	No	Dr. Winchester
Multivitamin	Take once daily	03/18/10	03/23/12	No	Dr. Vegat
Methylphenidate CD 40 mg	Take once daily	02/12/10	03/10/11	No	Dr. Winchester

Immunizations: Up to date; received annual influenza vaccine

Allergies: NKDA

PHYSICAL EXAM

BP 116/76 mm Hg | Pulse 90 beats per min
Temp 36.6°C (oral) | RR 14 breaths per min
Wt 41 kg | Ht 135 cm

General Appearance: Well-developed, well-nourished, overweight 11-year-old male in no apparent distress

Eyes: PERRLA, sclera clear, no conjunctival redness

Ears: TMs pearly, translucent, no erythema

Throat: Tonsils w/o erythema, tongue and uvula pink, no lesions

Neck: Neck supple, trachea midline, thyroid symmetrical, nontender, no palpable LNs

Lungs: Normal breath sounds, CTA all fields

Heart: RRR, no murmurs or gallops

Abdomen: Normal bowel sounds, no bruits, no tenderness or masses

Musculoskeletal: MS 5/5, no tenderness or atrophy

Skin: No abnormal lesions

Neurological: DTRs 2/4, sensory nerves intact

LABORATORY DATA[a]

BASIC METABOLIC PANEL		
Component	**Value**	**Range**
Glucose	88	60–110 mg/dL
BUN	12	8–19 mg/dL
Sodium	140	138–145 mmol/L
Potassium	4.3	3.4–5.2 mmol/L
Chloride	104	98–107 mmol/L
CO_2	26	20–28 mmol/L
Creatinine	0.6	0.2–0.7 mg/dL
Calcium	9.1	8.9–10.1 mg/dL
CBC WITH DIFF		
WBC count	7.84	4.5–13.0 x 10^3/μL
RBC count	4.82	4.00–5.20 million/μL
Hemoglobin	13.6	11.5–15.5 g/dL
Hematocrit	43.2	35.0% to 45.0%
MCV	86.8	77.0–95.0 fL
MCH	30.4	25.0–33.0 pg
MCHC	33.8	32.0–35.0 g/dL

BUN = blood urea nitrogen; CBC = complete blood count; CO_2 = carbon dioxide; MCH = mean corpuscular hemoglobin; MCHC = mean corpuscular hemoglobin concentration; MCV = mean corpuscular volume; RBC = red blood cell; WBC = white blood cell.

[a]All laboratory results are within normal limits.

DIAGNOSTIC TESTS

None provided

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PROBLEM LIST

- ❑ ADHD
- ❑ ODD

SELF-ASSESSMENT QUESTIONS

1. What is the subjective and objective evidence for the diagnosis of ADHD and ODD?
2. What risk and environmental factors may have contributed to ADHD in this patient?
3. How would you assess the current pharmacologic therapy for the patient?
4. What pharmacologic treatment would you recommend?
5. How would you counsel the patient and caregiver regarding the new medication?
6. The patient returns for follow-up after 8 weeks. At this point the guanfacine extended-release dose has been increased to 2 mg at bedtime. The patient's parents state that he has been feeling very dizzy, and he fainted yesterday. The patient's blood pressure is 90/50. Upon questioning the parents about how the medication is administered, they state that he usually eats dinner around 8 p.m. and takes the guanfacine dose after finishing his meal with dessert. What could be the cause of the adverse reaction? How will you instruct the patient to proceed?

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CASE 8.2
Autism Spectrum Disorder | Level 2

Jose Valdes and Jose A. Rey

LEARNING OBJECTIVES

1. Identify the presenting signs and symptoms of a child with autism spectrum disorder (ASD), and determine which symptoms would be the targets for pharmacotherapy.
2. Recommend nonpharmacologic and pharmacologic treatment options for the patient.
3. Devise treatment goals and outcomes for each of the identified healthcare problems presented in the case.
4. Detect a medication-related problem requiring intervention in the patient case.
5. Devise a follow-up plan for the patient case.

CHIEF COMPLAINT: "My daughter is becoming increasingly difficult to control!"

History of Present Illness: The patient is a 5-year-old Caucasian female with a previous history of ASD since age 2 and generalized tonic-clonic seizures diagnosed 6 months ago. She presented to the office with her mother stating that the patient "has become increasingly irritable in the mornings over the last week," which then leads to aggression (hitting her mother) and throwing tantrums throughout the day, demanding treats, and then banging her head against the door when she doesn't receive treats. "She insists on having me tie her shoes left to right before breakfast, otherwise she will hit me!" The patient's symptoms of irritability and sensory hypersensitivity were previously managed through physical and speech therapy, with modest improvements in behaviors and speech. However, according to her mother, her outbursts and tantrums are now interfering with her ability to participate in therapy, and she is unable to make any progress as a result.

The content of her speech is comparable to that of a 24-month old. Although she has had impairment in initiating conversations in the past, she recently has lost interest in maintaining and initiating a conversation with others. A daycare counselor has noted over the last month that the patient has been more socially withdrawn from other children and has had difficulty developing peer relationships. The patient spends much of her time sitting far away from others, focusing on the wheels of a toy bus in the classroom, and spinning them occasionally. Her emotions are blunted compared to others and she maintains a flat affect during social interactions. A casein-free and gluten-free diet was started a year ago with some additional improvement in behaviors, speech, and bowel habits (deceasing episodes of diarrhea); however, upon investigation the parents revealed that on days she behaved they would give her a treat and stray from the diet.

Review of Systems: Positive for bruising due to the head banging, hand tremor since starting valproic acid; no recent seizure activity according to mother

Past Medical History: Positive history of ASD since 2 years of age and seizure disorder (tonic-clonic seizures) diagnosed 6 months ago without any reported seizures since initiating treatment with valproic acid

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MEDICATION HISTORY

Medication	Sig	Start Date	End Date	Taking	Authorizing Provider
Valproic acid oral solution (250 mg/5 mL)	Give 10 mL po bid	6 mo ago		Yes	Dr. Doe

Past Surgical History: None

Social History: Lives with both parents and has no siblings; attends daycare and physical/speech therapy

Family History: Noncontributory

Diet: Gluten-free and casein-free

Immunizations: Up to date (not including seasonal allergy)

Allergies: NKDA

PHYSICAL EXAM

BP 97/69 mm Hg | Pulse 124 beats per min
Temp 99°F (oral) | RR 17 breaths per min
Wt 17.6 kg | Ht 92.9 cm | SpO_2 92%

General Appearance: Well developed, well nourished, uncooperative, and appears to be agitated

Eyes: Pupils reactive, EOMI; fundi normal, vision is grossly intact, bruising noted around the eyes

Ears: External auditory canals and tympanic membranes clear, hearing grossly intact

Throat: Oral cavity and pharynx normal; no inflammation, swelling, exudate, or lesions; teeth and gingiva in good general condition

Neck: No lymphadenopathy, supple with no meningismus, masses, or thyromegaly; trachea midline

Lungs: CTA bilaterally with good aeration, no retractions, no nasal flaring

Heart: Normal S1 and S2; no S3, S4, or murmurs; elevated heart rate, normal rhythm; there is no peripheral edema, cyanosis, or pallor; extremities are warm and well perfused; capillary refill is less than 2 seconds; no carotid bruits

Abdomen: Soft, nontender, +BS, nondistended, no HSM

Musculoskeletal: Adequately aligned spine; ROM intact spine and extremities; no joint erythema or tenderness; normal muscular development; normal gait

Skin: Flushed and sweating, no rashes, no petechiae, no purpura

Neurological: No gross deficits, strength 5 out of 5 upper and lower extremities; CN II–XII intact; strength and sensation symmetric and intact throughout; reflexes 2+ throughout; cerebellar testing normal; fine motor tremor noted in hands

DIAGNOSTIC TESTS

Childhood Autism Rating Scale (CARS) score of 38 performed 6 months ago

LABORATORY DATA

BASIC METABOLIC PANEL		
Component	**Value**	**Range**
Glucose	98	60–110 mg/dL
BUN	16	7–17 mg/dL
Sodium	142	138–145 mmol/L
Potassium	4.5	3.4–5.2 mmol/L
Chloride	102	98–107 mmol/L
CO_2	29	20–28 mmol/L
Anion gap	15	6–16 mmol/L
Creatinine	0.64	0.2–0.7 mg/dL
Calcium	8.9	8.9–10.1 mg/dL
CALCULATED OSMOLALITY		
Calc osmo	269	263–273 mOsm/kg
CBC WITH DIFF		
WBC count	5.4	4.5–11.0 x 10^3/µL
RBC count	4.8	4.30–5.10 million/µL
Hemoglobin	12.3	11.4–15.4 g/dL
Hematocrit	42%	36.0% to 49.0%

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LABORATORY DATA (cont'd)

CBC WITH DIFF (cont'd)		
MCV	85	78.0–98.0 fL
MCH	31	26.0–34.0 pg
MCHC	33	32.0–35.0 g/dL
RDW	13.7%	11.5% to 14.5%
Platelets	298	150–450 x 10^3/μL
MPV	11.9	9.4–12.4 fL
Neutrophils	67.2%	34.0% to 71.1%
Immature granulocytes	0.3%	0.0% to 0.5%
Neutrophils absolute	4.53	1.56–6.13 x 10^3/μL
Lymphocytes	45.8%	19.3% to 51.7%
Lymphocytes absolute	3.41	1.18–3.74 x 10^3/μL
Monocytes	12.4%	3.0% to 13.0%
Monocytes absolute	0.57	0.24–0.86 x 10^3/μL
Eosinophils	0.9%	0.7% to 5.8%
Eosinophils absolute	0.24	0.04–0.36 x 10^3/μL
Basophils	0.8%	0.1% to 1.2%
Basophils absolute	0.03	0.01–0.08 x 10^3/μL
VALPROIC ACID SERUM LEVEL		
VPA	139	50–100 mcg/mL
LIVER FUNCTION TEST		
AST	51	20–60 units/L
ALT	28	15–35 units/L
Ammonia	35	0–35 mmol/L

ALT = alanine transaminase; AST = aspartate aminotransferase; BUN = blood urea nitrogen; CBC = complete blood count; CO_2 = carbon dioxide; MCH = mean corpuscular hemoglobin; MCHC = mean corpuscular hemoglobin concentration; MCV = mean corpuscular volume; MPV = mean platelet volume; RBC = red blood cell; RDW = red cell distribution width; VPA = valproic acid; WBC = white blood cell.

PROBLEM LIST

- ❑ ASD with acute agitation and self-injurious behavior
- ❑ Seizure disorder (tonic-clonic)

SELF-ASSESSMENT QUESTIONS

1. What subjective and objective evidence supports the diagnosis of ASD?
2. Assess the nonpharmacologic and pharmacologic therapy for ASD and seizure disorder.
3. Develop an alternative plan to treat the patient's seizure disorder.
4. Discuss treatment outcomes and goals to using pharmacotherapy for reducing target symptoms of ASD as well as pharmacotherapy for the seizure disorder.
5. Develop a pharmacotherapeutic regimen for the treatment of the acute agitation/aggression and self-injurious behavior and discuss points to monitor.
6. What information would you provide to the caregivers and child to help manage symptoms of newly prescribed medications?

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CASE 8.3
Depression | Level 2

Kristen Lamberjack and Christine Prusa

LEARNING OBJECTIVES

1. Identify the signs and symptoms of depression in a pediatric patient.
2. Evaluate therapies used for the treatment of depression in pediatric patients.
3. Define treatment goals and outcomes.
4. Recommend pharmacologic and nonpharmacologic therapy for pediatric depression.
5. Recommend appropriate monitoring of pediatric patients initiating or changing antidepressant therapy.

CHIEF COMPLAINT: Anxiety and nervousness

History of Present Illness: The patient is a 13-year-old Caucasian female who presented for her first visit to the behavioral health physician after seeing her primary care physician (PCP) for the last 2 months for treatment of her depression. She was accompanied by her mother. She was referred by her PCP due to her lack of complete response to the prescribed antidepressant.

The patient began to experience symptoms of depression when she started 6th grade. She presented to her PCP 8 weeks ago with multiple symptoms including sadness on most days, irritability interfering with family and peer relationships, lack of motivation, low self-esteem, weight loss with loss of appetite, some guilt, difficulty falling asleep and waking/getting out of bed in the morning, and generalized fatigue/lack of energy. She also admitted to having some anxiety and nervousness. She was diagnosed by her PCP with depression at that time and started on fluoxetine 10 mg capsules once daily, and her dose was increased to 20 mg once daily 2 weeks ago. She has experienced moderate improvement in her depression symptoms; however, symptoms of anxiety and nervousness have been exaggerated since her dose was increased 2 weeks ago. She denies any suicidal or homicidal ideation.

DEPRESSION SCREEN

DEPRESSION/SADNESS: *Yes, but improving*

IRRITABILITY: *Yes, improving*

BOREDOM/APATHY: *Yes, apathy, improving*

ANHEDONIA: *Yes, improving*

SLEEP DISTURBANCE: *Yes, difficulty falling asleep and waking in the morning, some improvement*

INAPPROPRIATE GUILT: *No*

POOR ENERGY OR MOTIVATION: *Yes, improving*

POOR CONCENTRATION: *No*

APPETITE CHANGES: *Yes, improving*

CRYING EPISODES: *Yes, at night before bedtime, improving from baseline*

BIRTH HISTORY: *Noncontributory*

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MEDICATION HISTORY

Medication	Sig	Start Date	End Date	Taking	Authorizing Provider
Fluoxetine 10 mg capsule	1 capsule po daily	08/2013	09/2013	No	Dr. D. Press
Fluoxetine 20 mg capsule	1 capsule po daily	09/2013		Yes	Dr. D. Press
Loratadine 10 mg tablet	1 tablet po daily	04/2010		Yes	Dr. D. Press
Epinephrine 0.3 mg/0.3 mL IM autoinjector	Inject 0.3 mL IM prn for severe allergic reaction	03/2008		Yes	Dr. D. Press
Multivitamin	1 tablet daily	08/2012		Yes	Self-prescribed

IM = intramuscular.

Past Medical History: Seasonal allergies

Past Surgical History: Tympanostomy tubes, adenoids removed

Development History: No concerns

Social History: Lives with mother, stepfather, and two older sisters; attends 7th grade; no history of drug, tobacco, or alcohol use; no history of sexual behavior or abuse

Past Psychiatric History: No prior behavior problems; no history of bipolar disorder

Education History: No academic concerns or delays; earns primarily As/Bs; in the past year has been getting behind in school due to missed days (approximately 50 days)

Family History: Depression in mother, maternal grandmother, and aunt; mother responded well to paroxetine; no history of bipolar disorder

Immunizations: Up to date, including seasonal influenza vaccine

Allergies: Amoxicillin (rash), wheat flour, dust, peanuts (severe)

PHYSICAL EXAM

BP 104/63 mm Hg | Pulse 78 beats per min
Temp 98.7°F (oral) | RR 23 breaths per min
Wt 36 kg | Ht 148 cm

General Appearance: Alert, well appearing, no acute distress, exhibiting moderate anxiety/nervousness, small for age

HEENT: Head normocephalic; eyes, ears, nose, and throat clear

Lungs: Breath sounds clear and equal bilaterally, no respiratory distress, respirations easy and regular

Heart: Heart with regular rate and rhythm, no murmur

Abdomen: Soft without hepatosplenomegaly, no tenderness, no rebound or guarding, normal bowel sounds

Extremities: No deformity, full range of motion

Skin: Warm, dry with no significant birth marks or rashes

Neurological: Alert, normal tone

LABORATORY DATA

THYROID FUNCTION		
Component	**Value**	**Range**
TSH	3.5	0.7–5.4 micro-international units/mL
Free T_4	1.1	0.7–2.1 ng/dL
CBC WITH DIFF		
WBC count	5.8	4.5–11.0 x 10^3/µL
RBC count	4.39	4.30–5.10 million/µL
Hemoglobin	13.2	11.4–15.4 g/dL
Hematocrit	38.6	36.0% to 49.0%
MCV	80.6	78.0–98.0 fL
MCH	28.7	26.0–34.0 pg
MCHC	35.6	32.0–35.0 g/dL
RDW	12.1	11.5% to 14.5%
Platelets	238	150–450 x 10^3/µL
MPV	9.3	9.4–12.4 fL
Neutrophils	47	34.0% to 71.1%
Lymphocytes	45	19.3% to 51.7%

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LABORATORY DATA (cont'd)

CBC WITH DIFF (cont'd)		
Monocytes	7	3.0% to 13.0%
Eosinophils	1	0.7% to 5.8%
Basophils	0	0.1% to 1.2%

CBC = complete blood count; MCH = mean corpuscular hemoglobin; MCHC = mean corpuscular hemoglobin concentration; MCV = mean corpuscular volume; MPV = mean platelet volume; RBC = red blood cell; RDW = red cell distribution width; T_4 = thyroxine; TSH = thyroid-stimulating hormone; WBC = white blood cell.

PROBLEM LIST

- ❑ Depression
- ❑ Environmental allergies (well controlled)

SELF-ASSESSMENT QUESTIONS

1. What subjective and objective evidence supports the diagnosis of pediatric depression?
2. Assess the pharmacologic therapy for the patient's depression treatment and provide a treatment plan to optimize therapy.
3. Provide a follow-up schedule for pediatric patients initiating antidepressant therapy.
4. Discuss nonpharmacologic therapy and patient education for pediatric patients receiving antidepressants.
5. Discuss a treatment plan and goals for pediatric patients with depression.
6. Identify and provide a solution to the medication-related problem.

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CASE 8.4
Cerebral Palsy | Level 3

Mary Worthington

LEARNING OBJECTIVES

1. Identify the signs and symptoms of cerebral palsy and associated comorbidities in a child.
2. Assess the severity of spasticity for a pediatric patient and provide appropriate recommendations for treatment.
3. Recommend pharmacologic and nonpharmacologic therapy for drooling.
4. Formulate a treatment and monitoring plan for each of the identified healthcare problems presented in the patient case.
5. Identify a medication-related problem requiring intervention in the patient case.

CHIEF COMPLAINT: Pain in left leg

History of Present Illness: This is an 8-year-old white male with history of cerebral palsy and intellectual disability who presented to the emergency department with pain in the left lower extremity that has increased over the past week. He has generalized spasticity with the most prominent effects in the lower extremities. His mother reported over the past month, she has noted increased stiffness of both legs. Four months ago, he was seen for routine follow-up in the rehabilitation medicine clinic. Insertion of a baclofen pump was discussed, but it was decided to try clonazepam first. The clonazepam was discontinued a month later due to sedation. His oral baclofen dose was subsequently increased, but his mother is concerned about sedation with this new dose. In addition, she feels the stiffness worsens prior to baclofen dose. At baseline, the patient is unable to walk and is transported by wheelchair. The past week, when transferring to the wheelchair, he has become very uncomfortable and cries and grimaces with pain. He is also less comfortable in a sitting position. His mother has given ibuprofen, which provided only minimal relief.

Review of Systems: The patient is nonverbal, but his mother reports sedation that she contributes to the baclofen, drooling, constipation, and increased stiffness.

Birth History: Born at 37 weeks estimated gestational age

Past Medical History: Meningitis with brain abscess at 3 weeks of age due to *Citrobacter freundii;* cerebral palsy diagnosed at 1 year; spasticity; failure to thrive; gastroesophageal reflux disease

Past Surgical History: G-tube initially placed at 13 months, revised at 5 years

Development History: Communication—nonverbal, babbles, smiles, uses a communication board for five to six simple words; movement—transported by manual wheelchair, can maintain sitting posture (Gross Motor Function Classification System level V); social—enjoys music, attends special needs school 3 days per week

Social History: Lives with parents and 10-year-old sister

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MEDICATION HISTORY

Medication	Sig	Start Date	End Date	Taking	Authorizing Provider
Baclofen	1 mL (5 mg) per G-tube tid	6 yr PTA	3 yr PTA	No	Dr. Smith
	2 mL (10 mg) per G-tube tid	3 yr PTA	3 mo PTA	No	Dr. Smith
	4 mL (20 mg) per G-tube tid	3 mo PTA		Yes	Dr. Smith
Clonazepam	2.5 mL (0.25 mg) per G-tube bid	4 mo PTA	3 mo PTA	No	Dr. Smith
Ranitidine	3 mL (45 mg) per G-tube bid	3 yr PTA		Yes	Dr. Jones
Polyethylene glycol 3350	17 g per G-tube once a day	1 yr PTA		Yes	Dr. Smith
Multivitamin	7.5 mL per G-tube once a day	6 mo PTA		Yes	Dr. Jones
Calcium carbonate	5 mL (500 mg elemental calcium) per G-tube once a day	6 mo PTA		Yes	Dr. Jones
Cholecalciferol	2 mL (800 units) per G-tube once a day	6 mo PTA		Yes	Dr. Jones
Ibuprofen	10 mL (200 mg) per G-tube q 6 hr as needed	1 wk PTA		Yes	Self-prescribed

G-tube = gastrostomy tube; PTA = prior to admission.

Family History: Maternal grandmother with lupus; maternal aunt with autoimmune hepatitis

Diet: Peptamen Junior with Fiber®
240 mL G-tube 4 times daily, swallows with prompting, some thickened foods with assistance

Immunizations: Up to date including annual influenza vaccine

Allergies: NKDA

PHYSICAL EXAM

BP 95/55 mm Hg | Pulse 102 beats per min
Temp 36.5°C (oral) | RR 25 breaths per min
Wt 20 kg | Ht 115 cm | SpO_2 96% (RA)
FLACC score 7

General Appearance: Thin, chronically ill-appearing boy who is nonverbal moaning in discomfort, increased with movement of lower extremities

Eyes: PERRLA, EOM(I), sclera white, conjunctiva clear

Ears: TMs clear bilaterally

Throat: Poor dentition, excessive drooling with pooled saliva in mouth

Neck: No masses, no lymphadenopathy

Lungs: No retractions, breath sounds vesicular and equal

Heart: RRR, normal S1, S2, no m/r/g

Abdomen: Soft, no organomegaly, G-tube in place without leaking, decreased bowel sounds, stool palpable on left side

Musculoskeletal: Severe generalized spasticity in bilateral upper and lower extremities (Grade 3 for all 4 extremities on Modified Ashworth Scale) with contractures in all underlying major joints, wears ankle foot orthoses, no tenderness to palpation, no edema or swelling

Skin: Dry, intact, no pressure ulcers

Neurological: Truncal hypotonia, increased tone in extremities, 3 + deep tendon reflexes, symmetric, no pathological reflexes

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Account: s8879438

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LABORATORY DATA

BASIC METABOLIC PANEL		
Component	**Value**	**Range**
Glucose	85	60–110 mg/dL
BUN	15	8–19 mg/dL
Sodium	141	138–145 mmol/L
Potassium	4.3	3.4–5.2 mmol/L
Chloride	105	98–107 mmol/L
CO_2	23	20–28 mmol/L
Creatinine	0.3	0.2–0.7 mg/dL
Calcium	8.2	8.9–10.1 mg/dL
Phosphorous	4.0	3.8–5.5 mg/dL
LIVER FUNCTION TESTS		
Total protein	5.5	6–8 g/dL
Albumin	3.0	3.5–5 g/dL
Total bilirubin	0.3	0.2–1.3 mg/dL
ALT	35	10–30 units/L
AST	60	15–50 units/L
ALP	200	157–460 g/dL
CBC WITH DIFF		
WBC count	5.6	4.5–13.5 x 10^3/µL
RBC count	4.5	4.00–5.20 million/µL
Hemoglobin	10.5	11.5–15.5 g/dL
Hematocrit	34	35.0% to 45.0%
MCV	80	77.0–95.0 fL
MCH	26	25.0–33.0 pg
MCHC	32	32.0–35.0 g/dL
Platelets	260	150–450 x 10^3/µL

ALP = alkaline phosphatase; ALT = alanine transaminase; AST = aspartate aminotransferase; BUN = blood urea nitrogen; CBC = complete blood count; CO_2 = carbon dioxide; MCH = mean corpuscular hemoglobin; MCHC = mean corpuscular hemoglobin concentration; MCV = mean corpuscular volume; RBC = red blood cell; WBC = white blood cell.

DIAGNOSTIC TESTS: LEFT LEG, LATERAL SPINE, ABDOMINAL, AND HIP X-RAYS

No fractures seen; no hip dislocation or subluxation; osteopenia present; gas-diluted loops in the colon

MEDICATIONS AT ADMISSION

Ketorolac 10 mg IV now, then 10 mg IV q 6 hr prn pain, not to exceed 5 days treatment

Baclofen 40 mg (4 mL) per G-tube tid

Ranitidine 45 mg (3 mL) per G-tube bid

Polyethylene glycol 3350 17 g in water per G-tube once a day

Bisacodyl 10 mg PR now, then once a day prn constipation

Glycopyrrolate 0.4 mg (2 mL) per G-tube tid × 1 week, then increase to 0.8 mg (4 mL) per G-tube tid

Multivitamin 7.5 mL per G-tube once a day

Calcium carbonate 500 mg elemental calcium (5 mL) per G-tube once a day

Cholecalciferol 2 mL (800 units) per G-tube once a day

PROBLEM LIST

- ❑ Pain secondary to severe generalized spasticity related to cerebral palsy, consulting rehabilitation medicine services for baclofen pump
- ❑ GERD
- ❑ Constipation
- ❑ Drooling
- ❑ Low bone mineral density

SELF-ASSESSMENT QUESTIONS

1. Identify subjective and objective findings indicative of spasticity or other complications associated with cerebral palsy.
2. Evaluate the suitability of intrathecal baclofen therapy to treat the patient's increased spasticity.
3. Develop a plan to appropriately counsel the patient's parents on intrathecal baclofen therapy.
4. Assess the pharmacologic treatment of drooling in the patient and provide additional nonpharmacologic treatment strategies.
5. Provide recommendations to further assess the need for additional treatments for low bone mineral density.
6. Identify medication error and provide strategies for prevention.

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CASE 8.5
Seizure Disorders | Level 3

Jose Valdes and Jose A. Rey

LEARNING OBJECTIVES

1. Identify the presenting signs and symptoms of a seizure.
2. Differentiate between febrile and absence seizures.
3. Recommend nonpharmacologic and pharmacologic therapy for the patient in the case.
4. Devise treatment goals and outcomes for each of the identified healthcare problems presented in the patient case.
5. Detect a medication-related problem requiring intervention in the patient case.
6. Devise a follow-up plan for the patient in the case.

CHIEF COMPLAINT: "My son is acting strange, staring out into space"

History of Present Illness: The patient is a 4-year-old male with a history of simple febrile seizures. He presented with his mother to the emergency department with a low-grade fever, which started approximately 7 hours prior to arrival, s/p DTaP vaccine. His mother states she would have given her son "about two spoonfuls" of acetaminophen for the fever, but "the pharmacist told me his liver could go bad if I kept doing it so I stopped and took him to the doctor 2 weeks ago. The doctor said my son has a large liver with liver enzymes over 300 and that I shouldn't give him any acetaminophen products until his enzymes normalize on their own." His mother also states that the patient "has been acting strange lately." According to her report, the patient is "spacing out" for about a minute at a time and is unresponsive when it occurs. "He just stares out into space, and then snaps right out of it." His mother also states, "He has never been like this, but it's happened a few times since the first one!" The patient's mother is worried that it may worsen over time and that it can hurt her only child.

Review of Systems: According to the mother, just before the episodes occur he swallows then becomes unresponsive as his facial muscles and eyelids jerk briefly.

Past Medical History: Simple febrile seizures diagnosed at 18 months old and hepatomegaly diagnosed 2 weeks ago

Past Surgical History: None

Development History: Appropriate for age

Social History: Lives with both parents, only child, attends daycare

Family History: Father has epilepsy (type unspecified)

Diet: Average diet, no restrictions

Immunizations: Up to date (including annual influenza vaccination)

Allergies: NKDA

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MEDICATION HISTORY

Medication	Sig	Start Date	End Date	Taking	Authorizing Provider
Acetaminophen	400 mg po q 4 hr NTE 5 doses in 24 hr	Unknown	2 wk ago	No	OTC
Diazepam oral solution, 1 mg/mL	5 mg po q 8 hr at the first sign of a fever	At age 3		Yes	Dr. Smith

NTE = not to exceed; OTC = over the counter.

PHYSICAL EXAM

BP 113/75 mm Hg | Pulse 105 beats per min
Temp 101.7°F (oral) | RR 25 breaths per min
Wt 20.8 kg | Ht 93.9 cm | SpO_2 98% room air

General Appearance: Well developed, well nourished, alert, and cooperative and appears to be in no acute distress

Eyes: Pupils dilated, EOM(I); fundi normal, vision is grossly intact

Ears: External auditory canals and tympanic membranes clear, hearing grossly intact

Throat: Oral cavity and pharynx normal; no inflammation, swelling, exudate, or lesions; teeth and gingiva in good general condition

Neck: No lymphadenopathy, supple with no meningismus, masses, or thyromegaly; trachea midline

Lungs: CTA bilaterally with good aeration, no retractions, no nasal flaring

Heart: Normal S1 and S2; no S3, S4, or murmurs; RRR; there is no peripheral edema, cyanosis, or pallor; extremities are warm and well perfused; capillary refill is less than 2 seconds; no carotid bruits

Abdomen: Soft, nontender, +BS, nondistended, + HSM

Musculoskeletal: Adequately aligned spine; intact spine and extremities; no joint erythema or tenderness; normal muscular development; normal gait; normal ROM of all extremities

Skin: Flushed and sweating, no rashes, no petechiae, no purpura

Neurological: No gross deficits, strength 5 out of 5 upper and lower extremities; CN II-XII intact; strength and sensation symmetric and intact throughout; reflexes 2+ throughout; cerebellar testing normal

LABORATORY DATA

BASIC METABOLIC PANEL		
Component	**Value**	**Range**
Glucose	98	60–110 mg/dL
BUN	8	8–19 mg/dL
Sodium	140	138–145 mmol/L
Potassium	4	3.4–5.2 mmol/L
Chloride	100	98–107 mmol/L
CO_2	25	20–28 mmol/L
Anion gap	15	6–16 mmol/L
Creatinine	0.55	0.2–0.7 mg/dL
Calcium	9.2	8.7–9.8 mg/dL
CALCULATED OSMOLALITY		
Calc osmolality	277	275–290 mOsm/kg
CBC WITH DIFF		
WBC count	10.4	4.5–11.0 x 10^3/µL
RBC count	4.9	4.30–5.10 million/µL
Hemoglobin	12.8	11.4–15.4 g/dL
Hematocrit	42%	36.0% to 49.0%
MCV	85	78.0–98.0 fL
MCH	29	26.0–34.0 pg
MCHC	32.7	32.0–35.0 g/dL
RDW	12.4%	11.5% to 14.5%
Platelets	172	150–450 x 10^3/µL
MPV	10.2	9.4–12.4 fL
Neutrophils	59.7	34.0% to 71.1%
Immature granulocytes	0.2%	0.0% to 0.5%
Neutrophils absolute	2.94	1.56–6.13 x 10^3/µL
Lymphocytes	32.5%	19.3% to 51.7%
Lymphs absolute	2.47	1.18–3.74 x 10^3/µL
Monocytes	8.2	3.0% to 13.0%
Monocytes absolute	0.68	0.24–0.86 x 10^3/µL
Eosinophils	3.6%	0.7% to 5.8%
Eosinophils absolute	0.13	0.04–0.36 x 10^3/µL
Basophils	0.8%	0.1% to 1.2%
Basophils absolute	0.03	0.01–0.08 x 10^3/µL

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LABORATORY DATA (cont'd)

LIVER FUNCTION TESTS		
AST	67	20–60 units/dL
ALT	104	10–45 units/dL
Albumin	3.8	3.4–4.2 g/dL
Serum ammonia	27	0–35 mmol/L

ALT = alanine transaminase; AST = aspartate aminotransferase; BUN = blood urea nitrogen; CBC = complete blood count; CO_2 = c arbon dioxide; MCH = mean corpuscular hemoglobin; MCHC = mean corpuscular hemoglobin concentration; MCV = mean corpuscular volume; MPV = mean platelet volume; RBC = red blood cell; RDW = red cell distribution width; WBC = white blood cell.

DIAGNOSTIC TESTS

Electroencephalograph

Upon physical exam the patient is instructed to hyperventilate after being placed on an EEG. An EEG is performed at the bedside and reveals small, subtle spike and wave patterns at 3 Hz, generalized and symmetrical. Synchronous asymmetrical myoclonic arm jerks occur every 3 seconds with consistent eyelid twitching.

PROBLEM LIST

- ❑ Absence epilepsy
- ❑ Fever
- ❑ Hepatomegaly

SELF-ASSESSMENT QUESTIONS

1. What subjective and objective evidence supports the diagnosis of absence epilepsy?
2. Develop a nonpharmacologic and pharmacologic regimen for the treatment of the patient's epilepsy.
3. Assess which of the pharmacologic regimens to treat absence epilepsy would be best given the patient's hepatic impairment.
4. Describe the goal of pharmacotherapy and the desired outcomes in treating the patient's seizure disorder.
5. What information would you provide to the caregivers and child to help manage the symptoms?
6. Describe how to best manage the medication-related problem.

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CASE 9.1
Acute Otitis Media | Level 1

Dominic Chan and Jennifer Le

LEARNING OBJECTIVES

1. Identify the signs and symptoms commonly reported in a patient with acute otitis media (AOM).
2. Identify the risk factors associated with AOM.
3. Distinguish the appropriateness of the nonpharmacologic (i.e., initial observation) and pharmacologic approaches in treating AOM based on a patient's clinical presentation highly suggestive of AOM.
4. Formulate therapeutic goals and monitoring plans for the health issues provided in the problem list.
5. Detect and resolve a medication-related problem requiring intervention.

CHIEF COMPLAINT: Bilateral ear pain with fever

History of Present Illness: This is a 7-month-old previously healthy, full-term male infant who presented with 2 weeks of intermittent dry cough, congestion, and fever. Three days prior to presentation, the patient experienced fevers up to 104ºF, taken rectally, intermittently daily with slight improvement on ibuprofen. Overly anxious about the high fevers and increased fussiness, the parents presented their son to the pediatrician for antibiotic therapy. The parents denied any vomiting or altered mental status exhibited by the patient but reported their infant's increased bowel movements over the past 2 days. The parents also reported no change in feeding or appetite, and the patient had 14 wet diapers in the last 24 hours. The patient is observed tugging on both ears intermittently. Patient did not have any significant prior medical history.

Birth History: Born 39 weeks gestation via normal spontaneous vaginal delivery

Past Medical History: None

Past Surgical History: None

Development History: Not significant

Social History: Attends a daycare center; no siblings, pets at home, nor recent travel; no significant exposures

Family History: Father smokes 5 packs of cigarettes per day

Diet: Normal appetite; feeds on bottle with strong suck response; uses pacifier frequently

Immunizations: Up to date, including two doses of the current season's influenza vaccine

Allergies: No known drug or food allergies

PHYSICAL EXAM

BP 100/65 mm Hg | Pulse 153 beats per min | Temp 39.1ºC
RR 46 breaths per min | Wt 21 lb | Ht 74 cm

General Appearance: Appears WDWN; active with a strong cry; no apparent distress

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MEDICATION HISTORY

Medication	Sig	Taking	Authorizing Provider
Ibuprofen 100 mg/5 mL	25 mg (1.25 mL) po q 6 hr	Yes	Parent-initiated
Olive oil	1 drop au and po prn pain	Yes	Parent-initiated

EYES: Normal conjunctivae; pupils are equal, round, and reactive to light; no exhibition of discharge

EARS (BOTH RIGHT AND LEFT): External ear and pinna normal; no foreign bodies; tympanic membrane is abnormal, erythematous, and cloudy; a middle ear effusion is present; see Figure 1

NOSE: Nasal discharge present

THROAT: Oropharynx clear

NECK: Normal range of motion and supple

LUNGS: Normal effort; stridor, respiratory distress, wheezes, rhonchi, rales, and retraction are absent

HEART: Normal rate and regular rhythm

ABDOMEN: Soft without extension or tenderness

MUSCULOSKELETAL: No signs of injury and has free range of motion from all extremities

SKIN: Warm; no petechiae, purpura, or rash noted; no diaphoresis, cyanosis, mottling, jaundice or pallor

NEUROLOGICAL: Alert and exhibits normal muscle tone

LABORATORY DATA: None available

PROBLEM LIST

- ❑ Bilateral AOM

SELF-ASSESSMENT QUESTIONS

1. What is the subjective and objective evidence that supports the diagnosis of AOM?
2. What are the risk factors associated with AOM that are present in this patient?
3. Identify and resolve the medication-related problem.
4. Explain why this patient qualifies for initial observation or immediate pharmacologic treatment. If pharmacologic treatment is warranted, devise a plan for managing AOM.
5. What educational points should you provide to the anxious parents in optimizing the care of the patient's current and potential future infections?

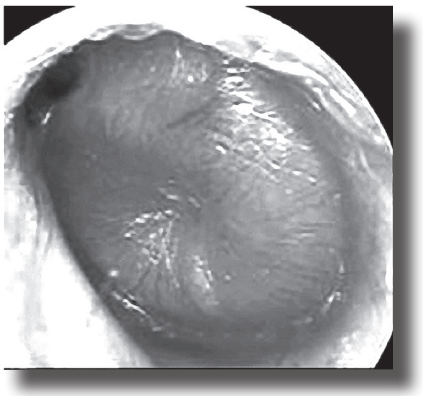

Figure 1. An otoscopy showing a tympanic membrane with moderate bulging.

Source: Subcommittee on Management of Acute Otitis Media. Diagnosis and management of acute otitis media. *Pediatrics*. 2013;131(3):e964–99.

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CASE 9.2
Skin and Skin Structure Infections | Level 1

Jennifer Le

LEARNING OBJECTIVES

1. Identify the signs and symptoms associated with skin and skin structure infections.
2. Differentiate between the different types of skin and skin structure infections—folliculitis, furuncles, carbuncles, erysipelas, cellulitis, impetigo, abscess, and necrotizing fasciitis—that can present in children.
3. Distinguish the appropriateness of the nonpharmacologic and pharmacologic approaches in managing different types of skin and skin structure infections.
4. Formulate therapeutic goals and monitoring plans for skin and skin structure infections.
5. Detect and resolve a medication-related problem requiring intervention.

CHIEF COMPLAINT: Leg swelling and redness

History of Present Illness: A 9-year-old Asian-Caucasian, previously healthy boy presented with left leg swelling and redness. His mother stated that he had a fever and some pain two nights ago, although she stated his temperature was 98°F. He had an abrasion on his left tibia 2 weeks ago. Mother said she had cleaned it but noticed that the pain and redness worsened. She took him to see his pediatrician today and subsequently to the emergency department due to the increasing redness and swelling.

Review of Systems: Some congestion for the past few days; otherwise, all systems reviewed negative except for those stated in his HPI

Birth History: Born full-term via repeat C-section; mother unsure of his birth weight but says there were no complications with pregnancy or delivery

Past Medical History: ADHD diagnosed 2 years ago; no hospitalizations or surgeries previously

Past Surgical History: None

Development History: Notable excessive temper tantrums and argumentative during pre-school; gets easily distracted and unwilling to complete chores

Social History: Lives with parents, 22-year-old brother, and 11-year-old sister; no special classes; no pets at home, recent travel, or tobacco exposure

Family History: Father—ADHD; no family history of skin lesions or boils

Diet: Normal diet with minimal vegetable intake; loss of appetite in past several weeks (not even eating his favorite foods like Kettle chips)

Immunizations: Up to date, including seasonal influenza vaccine

Allergies: NKDA

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MEDICATION HISTORY

Medication	Sig	Taking	Authorizing Provider
Dextroamphetamine/amphetamine (Adderall) XR	30 mg po once daily (drug initiated 6 mo ago; dose increased 2 wk ago)	Yes	Dr. Tran
Multivitamins	Two Gummi Vites po daily	Yes	Self-prescribed
Ibuprofen	200 mg po q 6 hr prn pain	Yes	Self-prescribed

PHYSICAL EXAM

BP 105/68 mm Hg | Pulse 95 beats per min
Temp 39.5°C | RR 22 breaths per min
Wt 50 lb (formerly 59 lb) | Ht 140 cm
SpO_2 99% | Pain 3/10 (numerical rating scale)

General Appearance: Appears thin and tired, although alert and oriented; recent weight loss (according to mother)

Eyes: Pupils—equal, round, and reactive to light

Ears: Tympanic membranes—gray, clear, and shiny bilaterally

Throat: Clear

Neck: Supple; no jugular vein distention

Lungs: Clear to auscultation bilaterally with good air exchange; no wheezes, rales, or rhonchi

Heart: Regular rate and rhythm with murmur ~3/6, heard loudest at the left sternal border; no thrill and no radiating to the back; no history of murmur according to mother

Abdomen: Soft, nontender, nondistended with positive bowel sounds; no hepatosplenomegaly

Musculoskeletal: Able to move his extremity; capillary refill <2 seconds; and peripheral pulses 2+

Skin: Has an abrasion under his nares with no erythema or drainage; left lower leg is red (nondemarcated), warm, tender, and swollen from knee to ankle; longest affected part measures 24 cm, leg circumference is 28 cm; circumferential redness from knee all the way down to ankle

Neurological: No deficits noted

LABORATORY DATA

BASIC METABOLIC PANEL		
Component	**Value**	**Range**
Glucose	111	60–110 mg/dL
BUN	7	7–17 mg/dL
Sodium	138	137–145 mmol/L
Potassium	4.8	3.6–5.0 mmol/L
Chloride	98	98–107 mmol/L
CO_2	29	22–30 mmol/L
Anion gap	11	6–16 mmol/L
Creatinine	0.4	0.60–1.00 mg/dL
Calcium	9.6	8.4–10.2 mg/dL
CALCULATED OSMOLALITY		
Calc osmo	270	263–273 mOsm/kg
CBC WITH DIFF		
WBC count	16.0	4.5–11.0 x 10^3/µL
RBC count	4.9	4.30–5.10 million/µL
Hemoglobin	13.1	11.4–15.4 g/dL
Hematocrit	38.3	36.0% to 49.0%
MCV	90	78.0–98.0 fL
MCH	31	26.0–34.0 pg
MCHC	33	32.0–35.0 g/dL
RDW	13.5	11.5% to 14.5%
Platelets	217	150–450 x 10^3/µL
MPV	10.0	9.4–12.4 fL
Neutrophils	79.1	34.0% to 71.1%
Immature granulocytes	0.5	0.0% to 0.5%
Lymphocytes	12.6	19.3% to 51.7%
Monocytes	5.5	3.0% to 13.0%
Eosinophils	1.6	0.7% to 5.8%
Basophils	1.2	0.1% to 1.2%
MICROBIOLOGY		
Blood culture	Pending	

BUN = blood urea nitrogen; CBC = complete blood count; CO_2 = carbon dioxide; MCH = mean corpuscular hemoglobin; MCHC = mean corpuscular hemoglobin concentration; MCV = mean corpuscular volume; MPV = mean platelet volume; RBC = red blood cell; RDW = red cell distribution width; WBC = white blood cell.

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DIAGNOSTIC TESTS

Chest x-ray showed a normal chest radiograph. X-rays of his femur (two views) revealed normal periosteal reaction. There is no evidence of gas or radiopaque foreign bodies in the skin structures. X-rays of his tibia and fibula showed no fractures or dislocations. There is no abnormal periosteal reaction. Overlying skin structures showed no abnormalities (no radiopaque foreign bodies and no gas). Impression is consistent with cellulitis.

MEDICATIONS AT ADMISSION

In the emergency department, patient received a normal saline bolus, cefazolin 400 mg intravenously, and morphine 2 mg intravenously. He was then transferred to the floor for hospital admission.

PROBLEM LIST

- ❑ Cellulitis
- ❑ ADHD

SELF-ASSESSMENT QUESTIONS

1. What is the subjective and objective evidence that supports the diagnosis of cellulitis and requirement for hospitalization?
2. Explain why the patient has cellulitis rather than erysipelas and necrotizing fasciitis by differentiating these types of skin and skin structure infections.
3. What are the most likely pathogens causing cellulitis in this patient?
4. Design an empiric antibiotic treatment regimen (including drug name, dose, frequency, and duration) that is appropriate for this patient.
5. Identify, resolve, and provide parent education for the medication-related problem.

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CASE 9.3
Parasitic Infections | Level 2

Heather L. Girand

LEARNING OBJECTIVES

1. Identify the presenting signs and symptoms of pinworm infestation and head lice in children.
2. Evaluate nonpharmacologic and over-the-counter (OTC) therapies used to treat pinworm infestation and head lice.
3. Develop a treatment plan for a child with pinworms and head lice.
4. Provide pertinent patient education for the parents regarding pinworm infestation and head lice management.
5. Detect a medication-related problem requiring intervention in the patient case.

CHIEF COMPLAINT: Perianal itching, erythema, and burning

History of Present Illness: A 6-year-old female presents to her pediatrician's office with a complaint of perianal itching for the past "few" days. Her mother reports that the child was preparing for her bath last night, and she noticed that her daughter was scratching her perianal area and stating, "my bottom hurts." The mother noted perianal erythema with areas near the anus that were "raw" from scratching. The child said that this area has been itchy for the past few days, but she was unable to determine exactly how long. That evening's bath did not relieve the symptoms, so the mother applied Cortizone-10 Plus cream to the area. Last night, the child awoke at 3 a.m. with intense itching and burning and was then unable to return to sleep. Her mother was concerned, so she brought her daughter to the pediatrician for evaluation. While waiting to be evaluated by the physician, the office nurse noted that the child was noticeably scratching behind her ears.

Review of Systems: Positive for perianal itching, erythema, and burning; fatigue

Birth History: Born at 41 weeks gestation via normal spontaneous vaginal delivery; no complications after birth; birth weight 3.6 kg

Past Medical History: Generally healthy; three episodes of sinusitis and one episode of acute otitis media since 1 year of age; mild eczema controlled with moisturizers

Past Surgical History: None

Development History: Normal with no concerns

Social History: Lives with mother, father, and 9-year-old sister; attends elementary school (first grade) and before/after school daycare; has one cat; recent history of travel on a 2-week camping trip with cousins of similar ages (shared sleeping quarters)

Family History: Asthma in father and sister; allergic rhinitis in mother

Diet: Normal for age

Immunizations: Up to date, including seasonal influenza vaccine

Allergies: Cefprozil—hives

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MEDICATION HISTORY

Medication	Sig	Start Date	End Date	Taking	Authorizing Provider
Cortisone 10-Plus Anti-Itch Creme (hydrocortisone 1%)	Apply to affected area 3–4 times daily as needed for itching	1 day PTA		Yes	Initiated by parent

PTA = prior to admission.

PHYSICAL EXAM

BP 110/68 mm Hg | Pulse 140 beats per min
Temp 37.5°C (oral) | RR 30 breaths per min
Wt 17 kg | Ht 115 cm | SpO_2 98% (RA)

General Appearance: Awake and alert; appears tired and uncomfortable

Head: Normal size; 50 + nits noted on the left postauricular side and midoccipital area of the head at the base of the hair shafts

Eyes: PERRLA; EOMs intact, conjunctiva clear; no eye discharge; red reflex present bilaterally

Ears: TMs clear bilaterally

Throat: Mucous membranes moist; no pharyngeal erythema or exudate

Neck: No lymphadenopathy; supple with full ROM

Lungs: Clear to auscultation

Heart: Tachycardic, normal S1/S2, no murmur

Abdomen: Soft, no distension; normal bowel sounds; no masses

Musculoskeletal: Normal range of motion; no edema

Skin: Warm, well perfused; capillary refill <2 seconds; perianal/perivaginal erythema; excoriated areas around anus with erythema spreading to groin

Neurological: Normal; 5/5 strength in upper and lower extremities; normal reflexes

LABORATORY DATA

None available

DIAGNOSTIC TESTS

Tape test = positive

MEDICATION PRESCRIBED BY PEDIATRICIAN AFTER OFFICE VISIT

Cephalexin 250 mg (250 mg/5 mL) orally 3 times daily

OTC therapy for pinworms and head lice (to be selected by parent based on availability at pharmacy)

PROBLEM LIST

- ❑ Pinworm infestation with secondary bacterial infection
- ❑ Head lice

SELF-ASSESSMENT QUESTIONS

1. What subjective and objective evidence supports the diagnoses of pinworm infestation and head lice in this patient?
2. Assess the use of hydrocortisone for this patient's symptoms related to pinworm infestation.
3. Develop a pharmacologic treatment plan for this patient's pinworm infestation and head lice.
4. Provide pertinent education for the parents regarding management of pinworm infestation and head lice.
5. Identify a medication-related problem in this patient and provide a recommendation for its management.

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CASE 9.4
Pneumonia | Level 2

Dominic Chan and Jennifer Le

LEARNING OBJECTIVES

1. Identify the signs and symptoms commonly reported in a patient with community-acquired pneumonia.
2. Differentiate between the two main types of pneumonia: community-acquired and healthcare-associated pneumonia.
3. Identify complications associated with community-acquired pneumonia and their implications in the etiology of disease and therapeutic management.
4. Formulate therapeutic goals and monitoring plans for community-acquired and healthcare-associated pneumonia.

CHIEF COMPLAINT: Persistent fevers

History of Present Illness: A 7-year-old girl with a history of hypothyroidism presented with persistent fevers (maximum temperature of 104.8°F) that initiated 6 days prior to hospital admission. In addition to fevers, her mother reported that over the past few days, the patient also experienced nonbloody, nonbilious emesis and constant midline epigastric pain, which initiated or worsened following meals. Her mother has been giving acetaminophen and ibuprofen around-the-clock coupled with cold compresses to treat her fever. Two days prior to admission, the patient developed a cough that progressed to labored breathing the following day. One day prior to admission, the patient also developed watery, nonbloody diarrhea without mucus. Since then, she has had three to four episodes of black diarrhea. Since admission, she has had a headache without notable vision changes. She has had decreased activity level, decreased oral intake, and a slight decrease in urine output. She exhibits no nasal congestion, no rashes, and no dysuria.

Review of Systems: All systems reviewed negative (except what is noted in her history of present illness)

Past Medical History: Graves disease, diagnosed 1½ years ago and status post radiotherapy

Past Surgical History: Thyroid ablation

Development History: Developmentally appropriate

Social History: Lives with her parents, 3-year-old sister, uncle, and one dog; no recent travel; no tobacco exposure; currently performing well in the 2nd grade

Family History: Father—hypertension; mother—unremarkable

Diet: Regular diet for age

Immunizations: Up to date including the influenza vaccine

Allergies: NKDA

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MEDICATION HISTORY

Medication	Sig	Taking	Authorizing Provider
Acetaminophen	320 mg po q 6 hr	Yes	Parent-initiated
Ibuprofen	200 mg po q 6 hr	Yes	Parent-initiated
Levothyroxine	75 mcg po daily	Yes	Dr. Smith

PHYSICAL EXAM

BP 106/64 mm Hg | Pulse 107 beats per min
Temp 39.3°C (oral) | RR 44 breaths per min
Wt 25.2 kg | Ht 131 cm | SpO_2 89%

General Appearance: Mild distress with nasal flaring; alert, nontoxic

Eyes: Extraocular movements intact; PERRLA

Ears: Left and right TMs clear

Throat: Oropharynx clear

Neck: Full range of motion; supple; tenderness to palpation over the anterior and posterior regions

Lungs: Clear to auscultation bilaterally; no retractions; taking shallow breaths

Heart: Tachycardia, regular rhythm; no murmurs

Abdomen: Soft, nondistended; tender to palpation diffusely, although more in the mid-epigastric region; no rebound tenderness; voluntary guarding is present

Musculoskeletal: 2+ pulses; capillary refill less than 2 seconds; no clubbing, cyanosis, or edema

Skin: No exudates or erythema

Neurological: Neurologically appropriate; nonfocal exam; cranial nerves II–XII intact

LABORATORY DATA

BASIC METABOLIC PANEL		
Component	**Value**	**Range**
Glucose	78	60–110 mg/dL
BUN	16	7–17 mg/dL
Sodium	138	137–145 mmol/L
Potassium	4	3.6–5.0 mmol/L
Chloride	100	98–107 mmol/L
CO_2	29	22–30 mmol/L
Anion gap	9	6–16 mmol/L
Creatinine	0.5	0.60–1.00 mg/dL
Calcium	7.9	8.4–10.2 mg/dL
CBC WITH DIFF		
WBC count	13.0	4.5–11.0 x 10^3/µL
RBC count	3.61	4.30–5.10 million/µL
Hemoglobin	10.4	11.4–15.4 g/dL
Hematocrit	32.1	36.0% to 49.0%
MCV	68.9	78.0–98.0 fL
MCH	28.8	26.0–34.0 pg
MCHC	32.4	32.0–35.0 g/dL
Platelets	227	150–450 x 10^3/µL
Neutrophils absolute	9.02	1.56–6.13 x 10^3/µL
Lymphocytes absolute	6.71	1.18–3.74 x 10^3/µL
Monocytes absolute	1.29	0.24–0.86 x 10^3/µL
Eosinophils absolute	1.17	0.04–0.36 x 10^3/µL
Basophils absolute	0.09	0.01–0.08 x 10^3/µL

BUN = blood urea nitrogen; CBC = complete blood count; CO_2 = carbon dioxide; MCH = mean corpuscular hemoglobin; MCHC = mean corpuscular hemoglobin concentration; MCV = mean corpuscular volume; RBC = red blood cell; WBC = white blood cell.

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DIAGNOSTIC TESTS

Chest x-ray shows moderate left lobar pleural effusion with a left opacity

PROBLEM LIST

- ❑ Complicated left lobe pneumonia
- ❑ Microcytic anemia
- ❑ Hypothyroidism post-thyroid ablation for Graves disease

SELF-ASSESSMENT QUESTIONS

1. What is the subjective and objective evidence that supports the diagnosis of community-acquired pneumonia?
2. What are the most likely pathogens causing community-acquired pneumonia in this patient, and what complication associated with pneumonia is presented?
3. Design a pharmacologic treatment regimen and monitoring plan for the treatment of complicated community-acquired pneumonia in this patient.
4. Identify and resolve the medication-related problem.

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CASE 9.5
Kawasaki Disease | Level 3

Jennifer Le

LEARNING OBJECTIVES

1. Identify the clinical and laboratory features commonly reported in a patient with Kawasaki disease.
2. Assess the risk factors and potential complications associated with Kawasaki disease.
3. Develop a comprehensive pharmacotherapeutic plan that incorporates drug therapy (including drug dose and duration of treatment), therapeutic goals, and monitoring parameters in a patient with Kawasaki disease.
4. Prevent and appropriately manage drug (including vaccines) and disease state interactions that may occur when using pharmacotherapies for Kawasaki disease.
5. Detect and resolve two medication-related problems requiring intervention.

CHIEF COMPLAINT: Fever (7 days), cough, and congestion (6 days)

History of Present Illness: In the emergency department with subsequent hospital admission, a 23-month-old Chinese girl presented with a 7-day history of daily fevers of 103°F. Six days prior to admission, the primary pediatrician diagnosed the patient with a viral syndrome due to the onset of cough and congestion. Four days prior to admission, the patient was seen by another emergency department where she was treated for possible dehydration and was sent home on an oral rehydration solution. During this time, dry lips and red tongue were noted. The pediatrician initiated amoxicillin due to concern for bacterial super infection 3 days prior to hospitalization. The patient continued to deteriorate with worsening respiratory symptoms and persistent fevers. Her parents noticed a maculopapular rash developing on her chest a day ago. They also reported diarrhea that initiated 4 days ago, was intensifying, appeared loose and nonbloody, and was occurring 15 times a day. In addition, her mother complained of the patient's unusual restlessness and fussiness over the past 2 days after she discontinued amoxicillin due to the rash. The patient has successfully completed a course of amoxicillin a year ago for an ear infection but did not develop the rash during that time.

Review of Systems: Positive fever, rash, upper respiratory illness, and nonbloody diarrhea; decreased oral intake and energy level; no emesis nor recent sick contacts; remaining 12-systems review was negative

Birth History: Vaginal delivery at 39 weeks gestation

Past Medical History: Full term, normal spontaneous vaginal delivery at 6 pounds 11 ounces; no prior hospitalization; history of chronic constipation; the patient stools once every 4 to 5 days and requires suppositories (per father)

Past Surgical History: None

Development History: Developmentally, the patient is meeting all milestones; speech and language development are appropriate for age; good gross motor coordination (sitting, walking, and running)

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MEDICATION HISTORY

Medication	Sig	Taking	Authorizing Provider
Diphenhydramine	6.25 mg po q 6 hr (2.5 mL of 12.5 mg/5 mL suspension)	Yes, ingested 4 doses over past 2 days	Parent-prescribed in response to rash
Amoxicillin	160 mg po q 8 hr for 7 days (4 mL of 200 mg/5 mL suspension)	No, discontinued by parents due to rash	Dr. Tran
Pedialyte liters	Drink 100 mL q 2 hr as needed for diarrhea	Yes	Dr. Escobar
Acetaminophen	160 mg (5 mL) po q 4–6 hr prn fever (Mother brought bottle of 160 mg/5 mL suspension)	Yes	Dr. Tran

Social History: Lives with parents; no pets; no exposure to smoking; grandmother who lives with patient traveled to China a month ago and brought some food back, but none of the foreign food was given to patient

Family History: Noncontributory; negative for chronic GI problems, such as ulcerative colitis and inflammatory bowel disease; no sudden infant death syndrome or congenital heart disease

Diet: Consumes grains, vegetables, fruits, and milk appropriate for age; does not like meat but eats it on occasion

Immunizations: Up to date except for varicella (not started yet) and PCV (received three of four injections); received seasonal influenza vaccine

Allergies: No known food allergies; rash to amoxicillin

PHYSICAL EXAM

BP 90/45 mm Hg | Pulse 177 beats per min RR 65 breaths per min | Temp 39.0°F axillary 98% SpO_2 | Wt 25 lb | Ht 82 cm

General Appearance: Nontoxic, no acute distress; crying on exam; fussy but consolable

Head: Normocephalic, atraumatic

Eyes: Positive red reflex; bilateral nonexudative redness

Nose: Nares are patent without gross rhinorrhea

Ears: Tympanic membranes intact, noninflamed

Mouth: Moist mucous membrane, with no marked erythema or exudates

Neck: Supple; no marked masses

Lungs: Clear to auscultation bilaterally; good equal air movement

Heart: Regular rate and rhythm; no murmurs, rubs, or gallops; normal S1 and S2

Abdomen: Soft, nontender, nondistended; positive bowel sounds; no masses appreciated

Musculoskeletal: Warm and well-perfused; swollen hands and feet noted

Skin: Diffuse blanching macular rash over abdomen; no vesicles, no lesions, and with some associated dry skin

Neurological: Positive Moro, suck, and grasp reflex; patient moving all four extremities equally and spontaneously

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LABORATORY DATA

BASIC METABOLIC PANEL		
Component	**Value**	**Range**
Glucose	113	60–110 mg/dL
BUN	7	7–17 mg/dL
Sodium	136	137–145 mmol/L
Potassium	4.4	3.6–5.0 mmol/L
Chloride	98	98–107 mmol/L
Bicarbonate	28	22–30 mmol/L
Anion gap	7	6–16 mmol/L
Creatinine	0.5	0.60–1.00 mg/dL
Calcium	9.1	8.4–10.2 mg/dL
CALCULATED OSMOLALITY		
Calculated osmolality	265	263–273 mOsm/kg
CBC WITH DIFF		
WBC count	26.7	4.5–11.0 x 10^3/µL
RBC count	3.9	4.30–5.10 million/µL
Hemoglobin	10.1	11.4–15.4 g/dL
Hematocrit	29.7	36.0% to 49.0%
MCV	92	78.0–98.0 fL
MCH	39	26.0–34.0 pg
MCHC	33	32.0–35.0 g/dL
RDW	12.5	11.5% to 14.5%
Platelets	877	150–450 x 10^3/µL
MPV	10.3	9.4–12.4 fL
Neutrophils	72	34.0% to 71.1%
Immature granulocytes	1	0.0% to 0.5%
Neutrophils absolute	6.50	1.56–6.13 x 10^3/µL
Lymphocytes	24	19.3% to 51.7%
Lymphocytes absolute	2.5	1.18–3.74 x 10^3/µL
Monocytes	7.0	3.0% to 13.0%
Monocytes absolute	0.52	0.24–0.86 x 10^3/µL
Eosinophils	1.5	0.7% to 5.8%
Eosinophils absolute	0.21	0.04–0.36 x 10^3/µL
Basophils	0.6	0.1% to 1.2%
Basophils absolute	0.03	0.01–0.08 x 10^3/µL
BLOOD, OTHER		
C-reactive protein	20.2	0–1.0 mg/dL
Erythrocyte sedimentation rate	56	3–13 mm/hr
Albumin	2.5	3.5–5.5 g/dL
Bilirubin, total	0.5	0–1.5 mg/dL
ALT	79	8–35 units/L
AST	56	15–46 units/L

URINALYSIS		
Color	Cloudy	
RBC	1	0–3/hpf
WBC	16	0–2/hpf
Glucose	Negative	Negative
Nitrite	Negative	Negative
Protein	Trace	0–20 mg/dL
Gram stain	Negative	Negative
MICROBIOLOGY		
Culture—blood	Pending	
Culture—stool	Pending	
Culture—urine	Pending	

ALT = alanine transaminase; AST = aspartate aminotransferase; BUN = blood urea nitrogen; CBC = complete blood count; MCH = mean corpuscular hemoglobin; MCHC = mean corpuscular hemoglobin concentration; MCV = mean corpuscular volume; MPV = mean platelet volume; RBC = red blood cell; RDW = red cell distribution width; WBC = white blood cell.

DIAGNOSTIC TESTS

Chest X-ray: Normal

Rapid Flu: Negative

Rapid RSV: Negative

Stool O&P Exam: Pending

MEDICATIONS AT ADMISSION: Normal saline bolus 250 mL × 1 dose

PROBLEM LIST

- ❑ Kawasaki disease

SELF-ASSESSMENT QUESTIONS

1. What subjective and objective evidence supports the diagnosis of Kawasaki disease?
2. What risk factors evident in this patient support the diagnosis of Kawasaki disease?

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3. What additional diagnostic test would you recommend to rule out the common complications of Kawasaki disease?
4. Recommend and explain the benefit(s) of pharmacologic treatment during hospitalization and at discharge. Provide the therapeutic goals and monitoring plans for this patient.
5. Identify, resolve, and provide parent education for two medication-related problems.
6. What educational points should you provide to the parents regarding the safe use of intravenous immune globulin, aspirin, and future vaccinations?

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CASE 9.6
Human Immunodeficiency Virus Infection | Level 3

Heather L. Girand

LEARNING OBJECTIVES

1. Identify presenting signs and symptoms of human immunodeficiency virus (HIV) in a young child.
2. Evaluate laboratory results for a young child with HIV and interpret the need for prophylaxis or treatment of infections based on those results.
3. Develop a treatment plan for a young child with HIV based on patient characteristics and laboratory results.
4. Establish treatment goals and outcomes for each of the identified healthcare problems presented in the patient case.
5. Detect a medication-related problem requiring intervention in the patient case.

CHIEF COMPLAINT: Fever, cough, difficulty breathing, and poor feeding

History of Present Illness: This is an 11-month-old male who developed a fever and cough 2 days ago. His foster mother reported that he felt "warm to the touch" a couple of days ago, but she did not take his temperature because she does not have a thermometer at home. He developed a dry cough that seemed to be getting more frequent and was interrupting his sleep. She gave him "cough medicine" last night, but she does not think that it helped. He has not been eating well for the past 3 days and refused all food and bottles this morning. This morning, his foster mother thought he was "struggling to breathe," so she took him to an urgent care center for evaluation. On arrival, his vital signs were rectal temperature 38.8°C, HR 166 beats per minute, RR 64 breaths per minute, BP 80/35 mm Hg, and oxygen saturation 84%. He was ill appearing with nasal flaring and intercostal and substernal retractions. A chest x-ray revealed bilateral diffuse parenchymal infiltrates. The urgent care center administered an IV bolus of normal saline (150 mL) and arranged for the child to be transferred to the hospital because of concern for respiratory failure. On arrival at the hospital, he was irritable, in moderate respiratory distress, and receiving supplemental oxygen via venturi mask (FiO_2 31%). A venous blood gas revealed pH 7.32, pCO_2 60 mm Hg, pO_2 55 mm Hg, and HCO_3 26 mmol/L. He was admitted to the general pediatric unit for treatment.

Review of Systems: Positive for fever, cough, difficulty breathing, and poor oral intake; no wheezing; no vomiting or diarrhea; no joint abnormalities or rash

Birth History: Born at estimated 38 weeks gestation via normal spontaneous vaginal delivery to a 17-year-old G1P1 mother who had no prenatal care; group B *Streptococcus* status of mother is unknown; no complications after birth; no hyperbilirubinemia; birth weight 2.82 kg

Past Medical History: Unknown; does not have a primary care physician but has an appointment with a pediatrician in 2 weeks

Past Surgical History: None

Development History: Delayed; not crawling or babbling

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MEDICATION HISTORY

Medication	Sig	Start Date	End Date	Taking	Authorizing Provider
Children's Robitussin Cough Long Acting (dextromethorphan)	7.5 mg (5 mL) q 8 hr as needed for cough (1 dose before bedtime on day of admission)	2 days PTA		Yes	Initiated by parent

PTA = prior to admission.

Social History: Has lived with foster parents for the past 2 weeks; biological mother's rights terminated for child neglect and arrest for drug dealing; no siblings; no daycare; no pets or smoke exposure currently

Family History: Unknown; biological mother known to have history of multiple sexual partners and sexually transmitted infections

Diet: Enfamil 6 ounces 3 times a day; minimal table foods

Immunizations: Received hepatitis B vaccine at birth; no other vaccines documented in state registry

Allergies: NKDA

PHYSICAL EXAM

BP 86/40 mm Hg | Pulse 162 beats per min
Temp 38.7°C (recta]) | RR 55 breaths per min
SpO_2 95% (on 31% venturi mask)
Wt 7.5 kg | Length 70 cm | HC 42.5 cm

General Appearance: Ill appearing, thin male infant in respiratory distress

Head: Small size; fontanelles closed

Eyes: Conjunctiva clear; no eye discharge; red reflex present bilaterally

Ears: Left TM clear; right TM obscured by cerumen

Throat: Mucous membranes dry; thrush noted on tongue

Neck: Shotty lymphadenopathy; no tenderness elicited on movement

Lungs: Bilateral basilar rales; no wheezing; intercostal and substernal retractions

Heart: Tachycardic, normal S1/S2, no murmur

Abdomen: Soft, nondistended; diminished bowel sounds; no masses

Musculoskeletal: Normal range of motion; no edema

Skin: Mottled, capillary refill 3 seconds; no petechiae; perineal erythema with satellite lesions

Neurological: Irritable but no gross deficits

LABORATORY DATA

BASIC METABOLIC PANEL		
Component	**Value**	**Range**
Glucose	130	60–110 mg/dL
BUN	17	2–13 mg/dL
Sodium	140	138–145 mmol/L
Potassium	4.1	3.5–5.9 mmol/L
Chloride	101	98–108 mmol/L
CO_2	15	19–30 mmol/L
Anion gap	24	6–16 mmol/L
Creatinine	0.5	0.2–0.4 mg/dL
Calcium	8.0	8.7–9.8 mg/dL
Albumin	3.0	3.4–4.2 g/dL
CBC WITH DIFF		
WBC count	3.2	6.0–17.5 $\times 10^3$/µL
RBC count	3.4	3.7–5.3 million/µL
Hemoglobin	9.8	10.5–13.5 g/dL
Hematocrit	29.4	33.0% to 39.0%
MCV	69.1	70.0–86.0 fL
MCH	22.0	23.0–31.0 pg
MCHC	29.1	30.0–36.0 g/dL
RDW	12.5	11.5% to 14.5%
Platelets	250	150–450 $\times 10^3$/µL
MPV	10.4	9.4–12.4 fL
Neutrophils	65	34.0% to 71.1%
Immature granulocytes	0	0.0% to 0.5%
Lymphocytes	10	19.3% to 51.7%
Monocytes	10	3.0% to 13.0%
Eosinophils	7.2	0.7% to 5.8%
Basophils	1.0	0.1% to 1.2%

BUN = blood urea nitrogen; CBC = complete blood count; CO_2 = carbon dioxide; MCH = mean corpuscular hemoglobin; MCHC = mean corpuscular hemoglobin concentration; MCV = mean corpuscular volume; MPV = mean platelet volume; RBC = red blood cell; RDW = red cell distribution width; WBC = white blood cell.

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DIAGNOSTIC TESTS

Blood culture negative

HIV rapid antibody test positive

HIV Western blot pending (later determined to be positive)

HIV quantitative RNA pending (later determined to be 190,000 copies/mL)

CD4 percentage pending (later determined to be 13%)

MEDICATIONS AT ADMISSION

D5W–½NS plus 20 mEq/L KCl at 45 mL/hr

Trimethoprim/sulfamethoxazole 37.5 mg (trimethoprim component) IV q 6 hr

Acetaminophen 96 mg po q 4–6 hr as needed for fever >38.6°C (101.5°F)

Dextromethorphan 7.5 mg po q 8 hr as needed for cough

PROBLEM LIST

- ❑ Presumed *Pneumocystis jiroveci* pneumonia
- ❑ HIV infection
- ❑ Dehydration
- ❑ Failure to thrive
- ❑ Thrush and candidal diaper rash
- ❑ Immunizations not up to date

SELF-ASSESSMENT QUESTIONS

1. What subjective and objective evidence supports the diagnosis of HIV in this child?
2. Assess the treatment plan for this patient's pneumonia and recommend changes to the regimen, if necessary.
3. If antiretroviral therapy will be initiated, what is an appropriate regimen for this child?
4. Provide a monitoring plan for this patient that takes into account current therapies and healthcare problems.
5. Discuss the medication-related problem in this patient and provide a recommendation for its management.

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CASE 9.7
Meningitis | Level 3

Heather L. Girand

LEARNING OBJECTIVES

1. Identify presenting signs and symptoms of bacterial meningitis in a young child.
2. Evaluate cerebrospinal fluid results obtained from a child with suspected meningitis.
3. Develop a treatment plan for bacterial meningitis based on patient characteristics and laboratory results.
4. Establish treatment goals and outcomes for treatment of bacterial meningitis.
5. Detect a medication-related problem in the patient case.

CHIEF COMPLAINT: Fever, irritability, vomiting, and lethargy

History of Present Illness: This is a 13-month-old female who developed a fever (39.1°C) and irritability 3 days ago. She had not been sleeping well and was waking up crying but consolable. Her mother reports that there was no associated cough, congestion, or rhinorrhea at the onset of this illness. Two days prior to admission, her mother took the child to a local urgent care clinic where she was diagnosed with acute otitis media (AOM) and prescribed amoxicillin (she has taken three doses) and acetaminophen for pain and fever (she received two doses yesterday). At 4:00 a.m. this morning, the child became extremely irritable, was inconsolable, and vomited twice. Since that time, she has been difficult to arouse and has not had anything to eat or drink. Her mother was concerned and brought the child to the emergency department.

Review of Systems: Positive for lethargy, irritability, and fever; no cough, wheezing, or SOB; positive for vomiting but no diarrhea; no joint swelling or erythema; no rash

Birth History: Born at 39 weeks gestation via normal spontaneous vaginal delivery; negative for group B *Streptococcus* status of mother; no complications after birth; no hyperbilirubinemia; birth weight 3.2 kg

Past Medical History: Healthy until current illness/recent diagnosis of AOM

Past Surgical History: None

Development History: Normal with no concerns

Social History: Lives with mother and father; attends daycare 2 days/week; one 4-year-old sister; no pets; no smoke exposure

Family History: Dyslipidemia—maternal grandfather and father; atopic dermatitis—mother and sister

Diet: Normal toddler diet; drinks cow's milk and eats table foods

Immunizations: No vaccines

Allergies: NKDA

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MEDICATION HISTORY

Medication	Sig	Start Date	End Date	Taking	Authorizing Provider
Amoxicillin	800 mg twice daily (10 mL of 400 mg/5 mL)	2 days PTA		Yes	Dr. Schultz
Acetaminophen	128 mg (4 mL of 160 mg/5 mL) q 4–6 hr prn	2 days PTA		Yes; as needed (2 doses yesterday)	Dr. Schultz

PTA = prior to admission.

PHYSICAL EXAM

BP 94/50 mm Hg | Pulse 160 beats per min
Temp 39.7°C (rectal) | RR 40 breaths per min
Wt 9.6 kg | Ht 75 cm | SpO_2 97% (RA)

General Appearance: Lethargic; high-pitched cry elicited during exam

Head: Normal shape; fontanelles closed

Eyes: Conjunctiva clear; no eye discharge; red reflex present bilaterally

Ears: Left middle ear effusion, no TM erythema or bulging; right ear canal obscured by cerumen

Throat: Mucous membranes moist; no pharyngeal erythema or exudate

Neck: No lymphadenopathy; unable to assess range of motion fully as movement elicited crying

Lungs: Clear to auscultation

Heart: Tachycardic, normal S1/S2, no murmur

Abdomen: Soft, no distension; normal bowel sounds; no masses

Musculoskeletal: Normal range of motion; no edema

Skin: Cool, mottled, capillary refill 3 seconds; no petechiae or rash

Neurological: Lethargic but irritable with exam; negative Kernig's and Brudzinski's sign

LABORATORY DATA

BASIC METABOLIC PANEL		
Component	**Value**	**Range**
Glucose	140	60–110 mg/dL
BUN	20	2–13 mg/dL
Sodium	143	138–145 mmol/L
Potassium	4.0	3.5–5.9 mmol/L
Chloride	103	98–108 mmol/L
CO_2	14	19–30 mmol/L
Anion gap	26	6–16 mmol/L
Creatinine	0.9	0.2–0.4 mg/dL
Calcium	8.8	8.7–9.8 mg/dL
CALCULATED OSMOLALITY		
Calc osmo	285	285–295 mOsm/kg
CBC WITH DIFF		
WBC count	22.3	6.0–17.5 x 10^3/µL
RBC count	4.1	3.1–4.5 million/µL
Hemoglobin	13.3	9.5–13.5 g/dL
Hematocrit	39.9	29.0% to 41.0%
MCV	99.4	74.0–108.0 fL
MCH	28.3	25.0–35.0 pg
MCHC	33.2	30.0–36.0 g/dL
RDW	13.5	11.5% to 14.5%
Platelets	300	150–450 x 10^3/µL
MPV	10.3	9.4–12.4 fL
Neutrophils	55	34.0% to 71.1%
Immature granulocytes	15	0.0% to 0.5%
Lymphocytes	20.5	19.3% to 51.7%
Monocytes	8	3.0% to 13.0%
Eosinophils	0.9	0.7% to 5.8%
Basophils	0.6	0.1% to 1.2%
Procalcitonin	20.1	<0.05 mcg/L
C-reactive protein	10.5	<0.8 mg/L

BUN = blood urea nitrogen; CBC = complete blood count; CO_2 = carbon dioxide; MCH = mean corpuscular hemoglobin; MCHC = mean corpuscular hemoglobin concentration; MCV = mean corpuscular volume; MPV = mean platelet volume; RBC = red blood cell; RDW = red cell distribution width; WBC = white blood cell.

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DIAGNOSTIC TESTS

LP RESULTS: Hazy yellow fluid; glucose 40 mg/dL; protein 640 mg/dL; WBC 3,000/mm^3 (88% neutrophils, 12% lymphocytes); RBC 150,000/mm^3

CSF GRAM STAIN: Negative

CSF CULTURE: Pending

BLOOD/URINE CULTURES: Pending

MEDICATIONS AT ADMISSION

NS 100 mL IV bolus, followed by D5W–½NS plus 20 mEq/L KCl at 40 mL/hr

Dexamethasone 1.4 mg IV q 6 hr

Acetaminophen 128 mg po q 4–6 hr as needed for fever >38.6°C (101.5°F)

PROBLEM LIST

- ❑ Meningitis
- ❑ Mild dehydration
- ❑ Immunizations not up to date
- ❑ Otitis media

SELF-ASSESSMENT QUESTIONS

1. What subjective and objective evidence supports the diagnosis of bacterial meningitis in this patient?
2. Develop a treatment plan for this patient's meningitis.
3. Assess the use of dexamethasone for this patient's meningitis.
4. Provide pertinent education for the parents regarding the prognosis and long-term outcomes for children with meningitis.
5. Discuss the medication-related problem in this patient and provide a recommendation for its management.

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CASE 10.1
Anemia | Level 1

Tracy M. Hagemann

LEARNING OBJECTIVES

1. Identify the presenting signs and symptoms of iron-deficiency anemia in a pediatric patient.
2. Evaluate the causes for iron-deficiency anemia for a pediatric patient and evaluate an appropriate treatment regimen.
3. Recommend both pharmacologic and nonpharmacologic therapies for pediatric iron-deficiency anemia.
4. Devise treatment goals and outcomes for each of the identified healthcare problems presented in the patient case.
5. Detect a medication-related problem requiring intervention in the patient case.

CHIEF COMPLAINT: None

History of Present Illness: A 14-month-old Hispanic male with a history of eczema on legs presented today to the pediatric clinic for a well-child checkup. Parents reported that he is a "fussy" eater and mostly drinks cow's milk and will only rarely eat solid food. He typically consumes a gallon of milk every other day. He was alert and active, although somewhat irritable today, and his parents report that he has not been sleeping well the past month. They deny bloody or black stools, jaundice, changes in bowel or urinary habits, or any breathing difficulties. He has been exhibiting some sneezing and a runny nose for which his mother has been giving him diphenhydramine. His growth and development have been normal.

Review of Systems: Normal appearing male, in no acute distress

Birth History: Term vaginal delivery

Past Medical History: Eczema (began at age 8 months)

Past Surgical History: None

Development History: No concerns

Social History: Lives with parents and two siblings, attends daycare; no smoking in the house, no pets

Family History: Noncontributory

Diet: Half-gallon of milk daily, occasional solid foods

Immunizations: Up to date, including seasonal influenza vaccine

Allergies: NKDA

PHYSICAL EXAM

BP 88/42 mm Hg | Pulse 125 beats per min | Temp 38°C (tympanic) | RR 28 breaths per min | Wt 12.2 kg | Ht 78 cm
SpO_2 98% (RA)

General Appearance: Alert, active

Eyes: PERRLA, clear

Ears: TM clear bilaterally

Throat: Normal, no exudate

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MEDICATION HISTORY

Medication	Sig	Start Date	End Date	Taking	Authorizing Provider
Diphenhydramine 12.5 mg/5 mL	2.5 mL (6.25 mg) daily at bedtime	1 mo ago		Yes	None—uses OTC
Eucerin®	Apply daily to affected areas	Age 6 mo		Yes	Dr. DeLeon

OTC = over the counter.

Neck: No lymphadenopathy, supple

Lungs: CTA bilaterally, no retractions

Heart: RRR, no murmur/gallop/clicks, no pedal edema

Abdomen: Soft, nontender, +BS, nondistended

Musculoskeletal: Well perfused, no edema

Skin: No rashes, no petechiae, no purpura, dry patches on lateral thigh, bilaterally

Neurological: No gross deficits, reflexes normal

LABORATORY DATA

BASIC METABOLIC PANEL		
Component	**Value**	**Range**
Glucose	103	60–110 mg/dL
BUN	6	7–17 mg/dL
Sodium	136	137–145 mmol/L
Potassium	4.5	3.6–5.0 mmol/L
Chloride	103	98–107 mmol/L
CO_2	23	22–30 mmol/L
Anion gap	10	6–16 mmol/L
Creatinine	0.36	0.60–1.00 mg/dL
Calcium	9.4	8.4–10.2 mg/dL
CALCULATED OSMOLALITY		
Calc osmo	279.9	263–273 mOsm/kg
CBC WITH DIFF		
WBC count	6.8	4.5–11.0 x 10^3/µL
RBC count	4.75	4.30–5.10 million/µL
Hemoglobin	8.4	11.4–15.4 g/dL
Hematocrit	28.2	36.0% to 49.0%
MCV	59.4	78.0–98.0 fL
MCH	17.7	26.0–34.0 pg
MCHC	29.8	32.0–35.0 g/dL
RDW	20	11.5% to 14.5%
Platelets	420	150–450 x 10^3/µL
MPV	9.8	9.4–12.4 fL

LABORATORY DATA (cont'd)

CBC WITH DIFF (cont'd)		
Neutrophils	54	34.0% to 71.1%
Immature granulocytes	0.4	0.0% to 0.5%
Neutrophils absolute	5.2	1.56–6.13 x 10^3/µL
Lymphocytes	22	19.3% to 51.7%
Lymphocytes absolute	2.6	1.18–3.74 x 10^3/µL
Monocytes	8	3.0% to 13.0%
Monocytes absolute	0.6	0.24–0.86 x 10^3/µL
Eosinophils	2	0.7% to 5.8%
Eosinophils absolute	0.12	0.04–0.36 x 10^3/µL
Basophils	1	0.1% to 1.2%
Basophils absolute	0.04	0.01–0.08 x 10^3/µL

BUN = blood urea nitrogen; CBC = complete blood count; CO_2 = carbon dioxide; MCH = mean corpuscular hemoglobin; MCHC = mean corpuscular hemoglobin concentration; MCV = mean corpuscular volume; MPV = mean platelet volume; RBC = red blood cell; RDW = red cell distribution width; WBC = white blood cell.

DIAGNOSTIC TESTS

None

PROBLEM LIST

- ❑ Microcytic anemia iron-deficiency
- ❑ Seasonal allergies
- ❑ At risk for overweight

SELF-ASSESSMENT QUESTIONS

1. What subjective and objective evidence supports the diagnosis of microcytic anemia?
2. Develop a nonpharmacologic and pharmacologic regimen for the treatment of iron-deficiency anemia in this patient.
3. Identify and propose a solution to the medication-related problem.

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4. What is the patient's current weight classification? What is the desired weight for this patient?
5. Devise the most pertinent patient education for the caregiver regarding both pharmacologic and nonpharmacologic therapy.

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CASE 10.2
Sickle Cell Disease | Level 2

Tracy M. Hagemann

LEARNING OBJECTIVES

1. Identify the presenting signs and symptoms of sickle cell pain crisis and acute chest syndrome in a pediatric patient.
2. Evaluate the causes for sickle cell complications, including pain crisis and acute chest syndrome for a pediatric patient and evaluate an appropriate treatment regimen.
3. Recommend both pharmacologic and nonpharmacologic therapies for SCD complications.
4. Devise treatment goals and outcomes for each of the identified healthcare problems presented in the patient case.
5. Detect a medication-related problem requiring intervention in the patient case.

CHIEF COMPLAINT: Left-sided chest pain × 1 day

History of Present Illness: A 15-year-old female with sickle cell disease (SCD) presented to the emergency department with left-sided chest pain, which started this afternoon, initially self-rated as 5/10 on a visual analog scale (VAS) and then pain gradually worsened to 10/10, sharp and throbbing in character, worse with movement, and when taking deep breaths. Patient reports that she took two hydrocodone 10 mg/acetaminophen 325 mg tablets, which did not improve her pain. She has also had a nonproductive cough for 4 days. She has a history of frequent admissions for sickle cell pain crisis in the past (averaging four per year).

Review of Systems: Pain 10/10; denies chills, denies sweats, denies anorexia, denies fatigue, denies malaise

Past Medical History: SCD—SS; multiple hospitalizations for pain crises

Past Surgical History: Cholecystectomy (5 years ago), port placement (4 years ago)

Development History: Normal development

Social History: Lives with mother, stepfather, brother, and sister; stepfather and brother smoke; in 10th grade; no pets at home

Family History: Cousin with SCD

Diet: Regular diet

Immunizations: In childhood, received DTaP, IPV, HepB, Hib, MMR, Var and HepA, received PPSV23 at age 2 years, and every year has received seasonal influenza vaccine (inactivated)

Allergies: NKDA

PHYSICAL EXAM

BP 121/71 mm Hg | Pulse 108 beats per min | Temp 39.1°C (oral) | RR 25 breaths per min | Wt 64 kg | Ht 167.6 cm SpO_2 88% RA | LMP 1 year ago

General Appearance: Awake, alert, in discomfort

Eyes: PERRLA, EOMs intact, conjunctiva clear

Ears: TMs clear, bilaterally

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MEDICATION HISTORY

Medication	Sig	Start Date	End Date	Taking	Authorizing Provider
Folic acid	1 mg po daily	01/06/2000		Yes	Dr. Parker
Ibuprofen	600 mg po q 6 hr prn pain	09/24/2011		Yes	Dr. Parker
Hydrocodone/acetaminophen 10/325	1 tablet po q 4–6 hr prn pain	09/24/2011		Yes	Dr. Parker
Hydroxyurea	1,000 mg po bid	11/22/2012		Yes	Dr. Parker
Medroxyprogesterone acetate	150 mg SC q 10–12 wk	11/22/2012		Yes	Dr. Parker

SC = subcutaneous.

Throat: Moist MM, throat normal, no exudate

Neck: No lymphadenopathy, supple

Lungs: No retractions, some decreased aeration on left

Heart: S1/S2 normal; I–II/VI systolic murmur best heard at LLSB; no rubs or gallops

Abdomen: Soft, nontender, +BS, nondistended; no hepatosplenomegaly appreciated

Musculoskeletal: Normal

Skin: No rashes, no petechiae, no purpura

Neurological: No gross deficits, normal reflexes; oriented × 3

LABORATORY DATA

BASIC METABOLIC PANEL		
Component	**Value**	**Range**
Glucose	109	60–110 mg/dL
BUN	10	7–17 mg/dL
Sodium	134	137–145 mmol/L
Potassium	4.2	3.6–5.0 mmol/L
Chloride	103	98–107 mmol/L
CO_2	22	22–30 mmol/L
Anion gap	9	6–16 mmol/L
Creatinine	0.29	0.60–1.00 mg/dL
Calcium	9.8	8.4–10.2 mg/dL
Total bilirubin	4.8	0.2–1.3 mg/dL
AST	38	14–36 units/L
ALT	36	9–52 units/L
Alkaline phosphatase	76	38–126 units/L
Albumin	4.6	3.5–5 g/dL
CBC WITH DIFF		
WBC count	18.7	4.5–11.0 × 10^3/μL
RBC count	2.37	4.30–5.10 million/μL
Hemoglobin	6.5	11.4–15.4 g/dL

LABORATORY DATA (cont'd)

CBC WITH DIFF (cont'd)		
Hematocrit	18.1	36.0% to 49.0%
MCV	101	78.0–98.0 fL
MCH	36.5	26.0–34.0 pg
MCHC	36.1	32.0–35.0 g/dL
RDW	21.6	11.5% to 14.5%
Platelets	158	150–450 × 10^3/μL
MPV	8.1	9.4–12.4 fL
Neutrophils	72	34.0% to 71.1%
Neutrophils absolute	13.5	1.56–6.13 × 10^3/μL
Lymphocytes	21	19.3% to 51.7%
Lymphocytes absolute	3.9	1.18–3.74 × 10^3/μL
Monocytes	3	3.0% to 13.0%
Monocytes absolute	0.6	0.24–0.86 × 10^3/μL
Eosinophils	4	0.7% to 5.8%
Eosinophils absolute	0.7	0.04–0.36 × 10^3/μL
Anisocytosis	2+	
Sickle cells	2+	
Retic count	19.0	0.45% to 2.0%
Absolute retic	450	18–158 K/mm^3

ALT = alanine transaminase; AST = aspartate aminotransferase; BUN = blood urea nitrogen; CBC = complete blood count; CO_2 = carbon dioxide; MCH = mean corpuscular hemoglobin; MCHC = mean corpuscular hemoglobin concentration; MCV = mean corpuscular volume; MPV = mean platelet volume; RBC = red blood cell; RDW = red cell distribution width; WBC = white blood cell.

MICROBIOLOGY

Source	Procedure—Status Growth
Throat	Group A *Streptococcus* culture—pending
Blood—peripheral	Blood culture—pending

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DIAGNOSTIC TESTS

CXR, no new infiltrates

MEDICATIONS AT ADMISSION

D5W–½NS at 100 mL/hr

Ketorolac 30 mg IV q 6 hr for 5 days

Morphine PCA 1.3 mg q 20 min with basal of 3.8 mg/hr for 1 hour limit of 7.7 mg

Ceftriaxone 2 g IV q 24 hr

Azithromycin 250 mg po daily

NS 0.9% flush q 8 hr and prn

Docusate 100 mg po tid

Folic acid 1 mg po daily

Hydroxyurea 1,000 mg po bid

Diphenhydramine 25 mg IV q 4–6 hr prn itching

pRBC transfusion 10 mL/kg over 4 hr

O_2 per NC to keep sats >92%

Incentive spirometry q 2 hr to prevent atelectasis

PROBLEM LIST

- ❑ Sickle cell pain crisis
- ❑ Acute chest syndrome suspected
- ❑ Anemia

SELF-ASSESSMENT QUESTIONS

1. What subjective and objective evidence supports the diagnosis of a sickle cell pain crisis and of acute chest syndrome?
2. Develop a pharmacologic regimen for the treatment of the pain crisis and the suspected acute chest syndrome in this patient.
3. Assess the pharmacologic therapy for each of the patient's problems.
4. Devise the most pertinent education for the patient and caregiver regarding both pharmacologic and nonpharmacologic therapy.
5. Why would this patient be using hydroxyurea? Assess the dose and describe how you should monitor her use of this drug.
6. Identify and propose a solution to a medication-related problem.

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CASE 10.3
Anticoagulation | Level 3

Brady S. Moffett

LEARNING OBJECTIVES

1. Identify the presenting signs and symptoms of an arterial thrombosis in a pediatric patient.
2. Recommend pharmacologic and nonpharmacologic therapy for arterial thrombosis.
3. Devise treatment goals and outcomes for each of the identified healthcare problems presented in the patient case.
4. Develop a strategy for patient education at discharge for outpatient therapy of an arterial thrombosis.

CHIEF COMPLAINT: Lack of perfusion to right arm

History of Present Illness: The patient is a 14-year-old male who presented to the pediatric emergency department (ED) with tachycardia, tachypnea, and fever (39.3°C). He arrived at the ED from home after sustaining a "mat burn" on his left lower thigh during wrestling practice at his school approximately 48 hours ago. The burn became inflamed, swollen, and began exuding pus within 24 hours. The patient attempted home therapy with frequent hydrogen peroxide washes and over the counter antibiotic cream with no improvement. On admission to the ED, the patient was administered normal saline fluid boluses (20 mL/kg × 3) and broad-spectrum antimicrobial coverage was initiated. The patient was admitted to the pediatric intensive care unit, continued to deteriorate, and began to experience mental status changes and became hypotensive. The patient was intubated and a peripherally inserted central catheter (PICC) was placed. An arterial line in the left radial artery was placed for invasive blood pressure monitoring. A continuous intravenous (IV) infusion of dopamine was initiated at 7.5 mcg/kg/min.

Approximately 48 hours after admission to the pediatric intensive care unit (PICU), the left hand was noticed to have decreased perfusion (grey, sluggish capillary refill, cold) and distal pulses in that hand were weak.

Birth History: Unremarkable

Past Medical History: None

Past Surgical History: None

Development History: Unremarkable and appropriate

Social History: Attends 8th grade; active in extracurricular sports activities

Family History: Lives with mother and father

Diet: Regular for age

Immunizations: Up to date, including influenza vaccine

Allergies: None

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Medication History: None, over-the-counter medication history unremarkable and included occasional ibuprofen

PHYSICAL EXAM

BP 100/70 mm Hg | Pulse 80 beats per min
Temp 36.5°C (oral) | RR 24 breaths per min
Wt 35 kg | Ht 98 cm | SpO_2 89% (RA)
Urine output (previous 24 hours) = 0.2 mL/kg/hr

General Appearance: Intubated and sedated male teenage child

Eyes: PERRLA, EOMs intact, conjunctiva clear

Ears: TM clear bilaterally

Throat: Moist MM, throat normal, no posterior pharynx exudate

Neck: No lymphadenopathy, supple with no meningismus, trachea midline

Lungs: Intubated patient, lungs CTA bilaterally with good aeration

Heart: RRR, no murmur/gallop/clicks, 2+ distal pulses, decreased pulses on the left arm

Abdomen: Soft, nontender, +BS, nondistended, no HSM

Musculoskeletal: Poorly perfused, capillary refill 3 to 4 seconds

Skin: No rashes, no petechiae, no purpura

Neurological: Sedated and intubated

LABORATORY DATA

BASIC METABOLIC PANEL		
Component	**Value**	**Range**
Glucose	140	60–110 mg/dL
BUN	32	7–17 mg/dL
Sodium	138	137–145 mmol/L
Potassium	4.8	3.6–5.0 mmol/L
Chloride	105	98–107 mmol/L
CO_2	28	22–30 mmol/L
Creatinine	1.6	0.60–1.00 mg/dL
Calcium	8.2	8.4–10.2 mg/dL

LABORATORY DATA (cont'd)

CBC WITH DIFF		
WBC count	2.86	4.5–11.0 ×10³/μL
RBC count	4.75	4.30–5.10 million/μL
Hemoglobin	11.8	11.4–15.4 g/dL
Hematocrit	38.0	36.0% to 49.0%
MCV	82.0	78.0–98.0 fL
MCH	28.7	26.0–34.0 pg
MCHC	32.7	32.0–35.0 g/dL
RDW	12.3	11.5% to 14.5%
Platelets	157	150–450 ×10³/μL
MPV	10.1	9.4–12.4 fL
Neutrophils	68.9	34.0% to 71.1%
Immature granulocytes	1.4	0.0% to 0.5%
Neutrophils absolute	8.3	1.56–6.13 ×10³/μL
Lymphocytes	56.7	19.3% to 51.7%
Lymphocytes absolute	3.9	1.18–3.74 ×10³/μL
Monocytes	12.6	3.0% to 13.0%
Monocytes absolute	0.57	0.24–0.86 ×10³/μL
Eosinophils	4.3	0.7% to 5.8%
Eosinophils absolute	0.31	0.04–0.36 ×10³/μL
Basophils	2.3	0.1% to 1.2%
Basophils absolute	0.11	0.01–0.08 ×10³/μL
Anticoagulation		
PT	21.1	15.6–17.5 sec
INR	1.5	1.1–1.3
aPTT	43	35–38 sec
Cultures		
Blood (×3)	No growth to date	
Urine	No growth to date	
Wound	No growth to date	
Spinal fluid	No growth to date	
Sputum	No growth to date	

aPTT = activated partial thromboplastin time; BUN = blood urea nitrogen; CBC = complete blood count; CO_2 = carbon dioxide; INR = international normalized ratio; MCH = mean corpuscular hemoglobin; MCHC = mean corpuscular hemoglobin concentration; MCV = mean corpuscular volume; MPV = mean platelet volume; PT = prothrombin time; RBC = red blood cell; RDW = red cell distribution width; WBC = white blood cell.

DIAGNOSTIC TESTS

Doppler ultrasound detects weak pulses.

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MEDICATIONS AT ADMISSION

Normal saline with 20 mEq/L of KCl IV 50 mL/hr

Dopamine IV 7.5 mcg/kg/min continuous infusion

Piperacillin/tazobactam 3.375 g IV q 6 hr (dose based on piperacillin content)

Vancomycin 525 mg (15 mg/kg/dose) IV q 6 hr

Fentanyl 2 mcg/kg/hr IV continuous infusion

Midazolam 0.05 mg/kg/hr IV continuous infusion

Ranitidine 35 mg (1 mg/kg/dose) IV q 6 hr

Heparin 1 unit/mL continuous infusion at 1 mL/hr through arterial line

Normal saline 10 mL flush q 8 hr IV through central line ports

PROBLEM LIST

- ❑ Arterial thrombosis
- ❑ Sepsis
- ❑ Acute kidney injury
- ❑ Respiratory failure and intubation
- ❑ Discharge/long-term therapy for arterial thrombosis

SELF-ASSESSMENT QUESTIONS

1. What is the subjective and objective evidence for the diagnosis of arterial thrombosis in this patient?
2. Devise a pharmacologic regimen for the treatment and prophylaxis of arterial thrombosis in a critically ill patient.
3. Determine the appropriate monitoring for the use of anticoagulation therapy in a critically ill patient with an arterial thrombosis.
4. Determine appropriate dosing of medications in a patient with sepsis and acute kidney injury.
5. Develop a strategy for continuation of outpatient anticoagulation therapy and to overcome common medication-related problems in the continuation of outpatient therapy.

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PART 2 | QUESTIONS & ANSWERS

2.1 Anaphylaxis—Level 1 117
2.2 Nutrition—Level 2 120
2.3 Toxicology—Level 3 126

3.1 Patent Ductus Arteriosus—Level 1 131
3.2 Hypertension—Level 2 133
3.3 Congenital Heart Defects—Level 3 136

4.1 Asthma—Level 1 139
4.2 Cystic Fibrosis—Level 2 143
4.3 Respiratory Distress Syndrome and Bronchopulmonary Dysplasia—Level 3 148

5.1 Constipation—Level 1 153
5.2 Crohn Disease—Level 2 157

6.1 Nephrotic Syndrome—Level 2 163
6.2 Acute Kidney Injury—Level 3 167

7.1 Type 2 Diabetes Mellitus—Level 1 173
7.2 Hypothyroidism—Level 2 176
7.3 Diabetic Ketoacidosis—Level 3 183

8.1 Attention Deficit Hyperactivity Disorder—Level 1 187
8.2 Autism Spectrum Disorder—Level 2 190
8.3 Depression—Level 2 195
8.4 Cerebral Palsy—Level 3 199
8.5 Seizure Disorders—Level 3 203

9.1 Acute Otitis Media—Level 1 209
9.2 Skin and Skin Structure Infections—Level 1 213
9.3 Parasitic Infections—Level 2 216
9.4 Pneumonia—Level 2 220
9.5 Kawasaki Disease—Level 3 223
9.6 Human Immunodeficiency Virus Infection—Level 3 227
9.7 Meningitis—Level 3 233

10.1 Anemia—Level 1 239
10.2 Sickle Cell Disease—Level 2 242
10.3 Anticoagulation—Level 3 247

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CASE 2.1
Anaphylaxis | Level 1

Nicole M. Even and Hanna Phan

1. What subjective and objective evidence supports the diagnosis of anaphylaxis?

SUBJECTIVE FINDINGS: After 5 minutes of receiving one dose of amoxicillin, the patient experienced lip swelling, complained of difficulty breathing, and developed a rash. She described the rash as itchy.

OBJECTIVE FINDINGS: On physical exam the patient's lips, tongue, and uvula are swollen; the rash is urticarial in nature with flushing and involvement of the neck, chest, and arms bilaterally. She is tachycardic with a heart rate of 160, tachypneic with a respiratory rate of 35, has positive dyspnea, and an oxygen saturation of 90% on room air. She also has positive nasal flaring, which is indicative of respiratory distress.

2. Develop a plan for anaphylaxis for the patient upon discharge.

The primary pharmacologic recommendation for anaphylaxis is the use of intramuscular (IM) epinephrine. This is the only medication that has been proven to prevent morbidity and mortality associated with anaphylaxis reactions in any patient population. Although it may seem logical to also give diphenhydramine or another antihistamine in addition to epinephrine, this is not recommended practice. The only medication recommended for an anaphylactic reaction is IM epinephrine. The recommended dose of epinephrine is 0.01 mg/kg of epinephrine (1:1000 [1 mg/mL] concentration) administered intramuscularly. Once given intramuscularly, epinephrine can take 5 to 10 minutes to start working and lasts for about an hour from a one-time dose. The dose can be repeated for one additional dose in 5 to 15 minutes after the first dose is given if the patient is still experiencing signs and symptoms of an anaphylactic reaction, which consist of hives, itching, flushing, swollen lips, swollen tongue, sensation of throat closure, choking, shortness of breath, wheeze, cough, syncope, dizziness, hypotension, and tachycardia. Giving too much epinephrine can result in cardiac arrhythmia and death. It is recommended that the dose be repeated only once unless the patient is in the care of medical professionals that deem additional doses are appropriate. The primary goals of treatment with epinephrine are to prevent any obstruction to airflow and to prevent the patient going into distributive shock. Prevention of airway obstruction and distributive shock are vital in an anaphylactic reaction in order to prevent the ultimate outcome of untreated anaphylaxis, which is death. Epinephrine helps prevent airway obstruction and distributive shock by increasing bronchodilation, decreasing

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the release of inflammatory mediators, and increasing vasoconstriction from its effects as an alpha-, beta-1, and beta-2 adrenergic agonist. For ease and expedited use, epinephrine is commercially available as autoinjectors in doses of 0.15 and 0.3 mg for outpatient use. The recommended dosing for the autoinjectors is dependent on weight-based ranges with patients weighing 10 to 25 kg receiving the 0.15 mg autoinjectors and patients weighing more than 25 kg receiving 0.3 mg autoinjectors. After the initial dose is administered, another dose can be repeated just one more time if needed 5 to 15 minutes after the initial dose if the patient is still experiencing signs and symptoms of anaphylaxis. For this patient, she should receive a prescription for the 0.15 mg autoinjector with a minimum quantity of two autoinjectors in the event the injection needs to be repeated in an anaphylactic situation. Please see question 4 below for more detailed instructions on autoinjector use and application.

3. Assess the pharmacologic therapy for the patient's acute otitis media. Why is this therapy no longer appropriate? What medication recommendation can you make to treat this infection?

The use of amoxicillin is no longer appropriate treatment based on the anaphylactic reaction the patient had after one dose. Based on the physical assessment, the patient had tympanic irritation and moderate bulging with symptoms for 7 days indicating this patient needs treatment for her acute otitis media. Due to the allergy to amoxicillin, this patient can no longer receive amoxicillin or amoxicillin–clavulanic acid. There is generally less than 10% cross-sensitivity with cephalosporins if the patient has a penicillin allergy. The cross-sensitivity of cephalosporins is based on the concept that the first-generation cephalosporins most resemble the beta-lactam ring structure found in penicillin. Studies demonstrate the cross-sensitivity is more closely related to the structure of the side chain on the beta-lactam ring of the cephalosporin structure. With the low chance for cross-sensitivity with penicillin allergy, it would be acceptable to give this patient a third- or fourth-generation cephalosporin, such as cefdinir. Based on the new allergy to amoxicillin and the guidelines for managing pediatric acute otitis media, this patient should be given cefdinir 322 mg (14 mg/kg) once daily or divided as twice daily to treat acute otitis media. The first dose should be administered in a physician's office so the patient can be observed for 1 to 2 hours to watch for an anaphylactic reaction. She should receive treatment for at least 5 days. If the physician is not comfortable giving cefdinir, then another option would be for the patient to be prescribed a combination of clindamycin 230 mg (10 mg/kg/dose) 3 times daily to treat the acute otitis media infection.

4. Devise the most pertinent patient education for the patient and caregiver.

The first step is for the patient and all caregivers to identify an anaphylactic reaction. Some signs and symptoms of an anaphylactic reaction include hives (raised, red, itchy patches of skin); swelling (in the eyes, lips, tongue, hands, feet, and throat); runny nose; trouble breathing; dizziness; or passing out. Once it is identified to be an anaphylactic reaction, the patient needs to receive a dose of epinephrine administered intramuscularly. Immediately after recognition of symptoms, call emergency medical services and have the patient lie down. Once the autoinjector is available, take it out of the box. Take off the carrier cap and remove the autoinjector from the carrier tube. Then remove the safety cap. Press the tip (opposite side from safety cap) firmly into the outer thigh regardless of clothing in the way. Hold the tip in place for at least 10 seconds. Education for the patient and all caregivers on how to use the epinephrine autoinjectors is key to its success in an anaphylactic situation. Under no circumstance should the patient try to stand up in an anaphylactic situation because this can make the patient go into distributive shock and cause empty ventricle syndrome. After the emergency medical services is contacted, the epinephrine from the autoinjector is administered, and the patient is lying down, the caregiver should wait for medical services to arrive and monitor the patient, watching for any signs and symptoms to indicate that the anaphylactic reaction is still occurring to determine if another dose of epinephrine needs to be administered (if avail-

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able). No more than two doses should be administered within a 15-minute time frame. After a dose of epinephrine, the patient may experience some adverse reactions from the medication such as feeling dizzy, anxious, or nauseous; having a headache; experiencing chest pain; or having a fast heartbeat.

The patient and all caregivers need to be familiar with the anaphylactic trigger the patient has and will need a written anaphylaxis emergency action plan prior to discharge from the hospital. The action plan needs to include instructions to identify and avoid any triggers, how to identify an anaphylactic reaction, how to use the epinephrine autoinjector, why it is used, when it needs to be used, and when an additional dose needs to be administered (if one is available). In addition, they need to notify medical assistance any time the autoinjector is used. It is not enough to simply show the patient and caregiver a picture of what the autoinjector looks like. They need to be taught with a practice device on exactly what it looks like, how it is supplied, and how to give it in an anaphylactic situation. Although there are instructions on the side of the autoinjector, the patient and caregiver need to know how to use it well before actually being in the situation where they need to give it. The action plan also needs to be given to any additional caregivers that may help take care of the patient (e.g., school nurse, sports coaches), and the patient needs to keep an autoinjector with them at all times.

5. What precautions need to be given as a result of this anaphylactic reaction regarding the patient's medical care communication in the future?

The patient experienced an anaphylactic reaction to a single dose of amoxicillin. This reaction needs to be documented in the patient's permanent medical record, and the patient and caregivers need to inform any medical personnel of this reaction any time the patient has an interaction with medical personnel that would require antibiotic treatment. It is also recommended that the patient wear a medical alert bracelet that has this information on it in case this occurs again when a caregiver is not present. Due to the patient having an anaphylactic reaction to amoxicillin, she should not receive any medications that contain amoxicillin or any cephalosporins that would have the possibility of having cross-sensitivity to penicillin (i.e., first-generation cephalosporins or cephalosporins with a similar side chain).

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CASE 2.2
Nutrition | Level 2

Sandra Benavides

1. What is the subjective and objective evidence of dehydration in this patient?

SUBJECTIVE FINDINGS: Decreased wet diapers and lethargy

OBJECTIVE FINDINGS: Decreased blood pressure, increased heart rate, decreased weight, lethargic, sunken fontanel, eyes sunken, dry mucous membranes, delayed capillary refill and skin turgor; BUN/SCr ratio of 35

2. Determine the severity of dehydration and devise a treatment for the patient in the case.

The patient has moderate dehydration as the patient has lost approximately 8% of her body weight and is presenting with symptoms consistent for moderate dehydration in infants including tachycardia, low systolic blood pressure, decreased urine output, dry mucous membranes, sunken fontanel and eyes, and delayed skin turgor. The percent dehydration is determined by amount of body weight loss from before the illness to after the illness [$(7.2 - 6.6)/7.2 \times 100 = 8.3\%$]. The likely cause for the patient's dehydration is decreased oral intake and increased output with vomiting over the past week.

Treatment of dehydration is divided into emergency management and replacement management. The type of dehydration is also important in the determination of IVF to administer. In this patient, her sodium is in the normal range, which indicates isonatremic dehydration indicating the amount of sodium and water loss is approximately the same. This is the most common type of dehydration in children.

Initially, if the patient is hemodynamically unstable, the administration of an intravenous bolus is necessary, which would be considered the emergency management of the patient. A bolus of 20 mL/kg (based on the pre-illness weight, in this patient would be 144 mL over 30 minutes) of normal saline or lactated Ringer's solution should be administered immediately. The use of normal saline is an appropriate choice in this patient. If the patient remains hemodynamically unstable, an additional bolus at the same dose can be repeated until circulation is stable. If a second bolus of 20 mL/kg is ineffective, 40 mL/kg can be administered. The goal of therapy with the IVF bolus is to restore intravascular volume in order to prevent hypoperfusion and damage to the organs.

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After the initial IVF bolus(es), the replacement management begins. The remaining water deficits are calculated. In this patient, the total water deficit is 598 mL. Fluid deficit (in liters) is determined by multiplying the percent dehydration times the pre-illness weight. It is also necessary to calculate the maintenance intravenous fluid (MIVF), which equals 720 mL when using the Holliday-Segar method (100 mL/kg/day for first 10 kg based on the pre-illness weight). The total amount of fluid to administer over the next 24 hours is 1,318 mL. The rate of replacement should be ½ of remaining deficit (598 mL – 144 mL administered as bolus = 454 mL divided by two is equal to 227 mL) and ⅓ MIVF (720 mL divided by three is equal to 240 mL) in the first 8 hours (227 mL + 240 mL = 467 mL over 8 hours) and the remaining over the next 16 hours (227 mL + 480 mL = 707 mL over 16 hours), respectively. The rate of infusion of normal saline should be 58.4 mL/hr × 8 hours, followed by 44.2 mL/hr × 16 hours. This should be administered until the PN arrives.

3. Develop an initial PN formula with the same caloric content as her current dietary intake for administration via a peripheral line.

a. Determine the fluid requirements for the infant: Based on the Holliday-Segar formula, the MIVF is 720 mL per day.

b. Determine the caloric needs: The infant receives 1,032 mL (equivalent to 35 ounces) of formula with 20 kcal/ounce, which provides 700 kcal per day. The caloric requirements for an infant between 1 to 12 months are 80 to 105 kcal/kg. This patient had been receiving approximately 97 kcal/kg, which falls into the recommended caloric intake for the patient's age.

c. Calculate the rate of infusion: The total volume to be infused over 24 hours is 720 mL minus the volume of the intravenous lipids (see below, total of 108 mL). Therefore, the appropriate infusion rate equals 25.5 mL/hr (612 mL over 24 hours).

d. Calculate carbohydrate requirements: Because the patient only has a peripheral line, the concentration of carbohydrates cannot exceed 12.5%. The patient can start on a 10% dextrose formula to determine tolerance and advanced at 5 g/kg per day. With the insertion of a central line, the dextrose concentrations can be higher. The caloric intake can be determined by calculating the total number of grams in 24 hours (612 mL of 10% solution is equal to 61.2 g). Each gram of carbohydrate provides 3.4 kcal. The total caloric intake in this patient from carbohydrates is 208 kcal in 24 hours for a 10% solution.

e. Calculate the protein intake: The patient is currently receiving 21.7 g of protein through her enteral nutrition (each 100 calories of formula has 3.1 g of protein). This is equal to 3 g/kg of protein, which falls into the recommended protein intake of 2 to 3 g/kg for infants. As such, it is reasonable to provide the same amount of protein in the PN. The total caloric intake in this patient from protein is 87 kcal in 24 hours.

f. Calculate the fat intake: The patient is currently receiving 32.9 g of fat through her enteral nutrition (each 100 cal of formula has 4.7 g of fat). This is equal to 4.6 g/kg, which exceeds the recommended range of 1 to 3 g/kg for healthy children. However, this patient may require greater fat caloric intake in her TPN to ensure adequate growth and development due to underlying health conditions (i.e., cardiac abnormalities). The overall caloric percentage obtained from fat should be calculated to ensure it does not exceed 50%. It is reasonable to decrease the total grams to 21.6 g (= 3 g/kg × 7.2 kg) to determine tolerance to the lipid emulsion. The lipid emulsion is typically infused over 12 hours through a separate line. In this patient, administration of 9 mL/hr for 12 hours equals 3 g/kg/day. The total caloric intake in this patient from fat is 216 kcal in 12 hours (21.6 g × 10 kcal/g of 20% lipid emulsion). This is less than 50% of total caloric intake (42%).

g. Total caloric intake: The patient is receiving 511 kcal per day from TPN, which is below her required caloric intake. Increasing her dextrose to 12.5% would provide 260 kcal from carbohydrates and

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563 kcal per day. However, increasing the dextrose concentration also increases the osmolarity of the solution. In pediatric patients, the osmolarity of the solution should not exceed 900 mOsm/L. With placement of a central line, the patient should be able to receive 100% of her caloric needs.

h. Electrolyte requirements: Electrolyte requirements can be estimated based on the daily maintenance requirements for a specific age as shown in **Table 1**. An alternative approach is to determine the current intake of each electrolyte and order that amount for the PN. EleCare® contains 2 mEq of sodium per 100 calories of formula (obtained from the nutrition information for the formula). Therefore, the patient receives 14 mEq of sodium per day (approximately 2 mEq/kg/day). Because her sodium was WNL, the PN may contain the same amount. The salt form to include is decided by the acid-base status of the patient and the phosphate requirement. In this case, because she has no current acid-base disorders, the 2 mEq can be administered as sodium acetate. For the potassium, the patient currently receives 27.3 mEq/day (3.8 mEq/kg/day). Her current intake of chloride is approximately 12 mEq/day (1.7 mEq/kg/day). The 4 mEq of potassium can be divided into 2 mEq of potassium chloride and 2 mEq of potassium acetate. That will supply the same amount of chloride she has been receiving from enteral feedings. The patient is currently receiving 41 mEq/day of calcium (5.7 mEq/kg/day). However, the amount of calcium that can be administered through PN is limited by osmolarity and phosphorus concentrations. In this patient, because she has only a peripheral line, it is reasonable to provide a lower amount of calcium gluconate and increase once she has a central line. Additionally, because of limitations in osmolarity and solubility, phosphorus can be added once a central line is placed. The optimal ratio of calcium:phosphorus is 1.7:1 throughout the first year of life for optimal bone mineralization. The patient is currently receiving 5.8 mEq/day of magnesium (0.8 mEq/kg/day). Although this amount exceeds the recommendations for infants and children, it can be added in this quantity because it is what she has been receiving in her enteral formula. However, a serum magnesium concentration should be obtained to ensure it is in normal range.

i. Add vitamins and trace elements: Typically, for short-term PN, a calculated dose of commercially available vitamins and minerals are added to the peripheral nutrition. The dose for the pediatric vitamins is 2 mL/kg (max of 5 mL). In this patient, she would receive the 5 mL. For trace elements, the dose is 0.2 mL/kg (max of 5 mg). This patient would receive 1.4 mL each day. It is important to be cognizant of various formulations of vitamins and trace elements.

TABLE 1: Estimated Daily Electrolyte Requirements

Electrolyte	Neonates	Infants/Children	Adolescents
Sodium	2–5 mEq/kg/day	2–6 mEq/kg/day	2–4 mEq/kg/day
Potassium	1–4 mEq/kg/day	2–4 mEq/kg/day	1–2 mEq/kg/day
Chloride	1–5 mEq/kg/day	2–5 mEq/kg/day	2–4 mEq/kg/day
Calcium gluconate	3–4 mEq/kg/day	1–2.5 mEq/kg/day	10–20 mEq/day
Phosphorus	1–2 mmol/kg	0.5–1 mmol/kg	10–40 mmol/day
Magnesium	0.3–0.5 mEq/kg/day	0.3–0.5 mEq/kg/day	10–30 mEq/day

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Typically, neonate, pediatric, and adult-specific formulas are available and vary in the concentration of the vitamins and trace elements. Unfortunately, drug shortages of trace elements have occurred frequently necessitating the addition of the individual trace elements or imported products from other countries. In such scenarios, it is critical to carefully select and dose the appropriate components and pay special attention to the concentrations of each additive.

Figure 1 illustrates an example of a PN order form.

4. Develop a monitoring plan for PN therapy.

Extensive monitoring is required for patients on PN. Body weight should be obtained daily to ensure adequate nutrition support for growth and development. Vital signs, including temperature or signs of infection, should be monitored daily as well. Ideally, prior to the initiation of PN, electrolytes (including phosphorus and magnesium), renal and liver function tests, triglycerides, and a CBC should be obtained. During the initiation of PN, a daily CMP is ideal to monitor adjustments in the PN. If the laboratory analysis is not obtained prior to initiation of PN, it should be obtained within 72 hours. Once a patient is stabilized, the frequency can decrease to once weekly. Also important to monitor are "ins and outs" to assess fluid status daily.

Once the patient is able to tolerate enteral nutrition, the PN should be discontinued and the enteral GI feeds restarted.

5. List potential complications of PN.

Complications of PN can be categorized into mechanical, infectious, or metabolic. *Mechanical complications* include problems with the pump or tubing. Catheter-related problems include hematoma formation, pneumothorax, arterial puncture, air embolism, venous thrombosis, and catheter occlusions. *Infectious complications* include contamination of line during placement or due to poor catheter care or from another source of infection.

Metabolic complications are extensive. Hyperglycemia or glucosuria can occur if the dextrose infusion rate is too high. Rebound hypoglycemia can occur if the PN is stopped abruptly. Alterations in any electrolyte can occur due to excess or inadequate administration or other disease processes (e.g., hyperkalemia due to renal impairment, hypophosphatemia due to alkalosis). Cholestatic jaundice is a complication of long-term PN and is best avoided by initiating enteral nutrition as soon as possible. Other complications include prerenal azotemia, hypercapnia, hyperchloremic acidosis, and metabolic alkalosis.

6. Identify the medication-related problem and propose a solution.

The patient is experiencing constipation as evidenced by the one bowel movement in the last week. The change that occurred in that time is the addition of glycopyrrolate, an anticholinergic agent, at the higher end of the dosing range. Typically, the medication is initiated at the lower recommended dose and titrated up to effect. The recommended dose to minimize excess salivation is 40 to 100 mcg/kg/dose 3 to 4 times daily. The patient was prescribed 0.7 mg 4 times daily, which is equal to 100 mcg/kg/dose. A more appropriate initial dose is 0.3 mg 4 times daily. Adverse reactions to glycopyrrolate include tachycardia; drowsiness; insomnia; constipation; dry eyes, mouth, and skin; and urinary retention. In the patient, the high starting dose may have contributed to the constipation. The dose should be decreased to 0.3 mg per G-tube 4 times daily.

Secondarily, the parent should be reminded to obtain a second dose of the influenza vaccine after 2 weeks. In children less than 9 years of age, two doses are required the first year the influenza vaccine is administered: the first dose serves to prime the immune system, and the second dose offers protection. Incomplete vaccination (i.e., only one dose) will not confer protection to the child. After the initial two doses in one season, only one dose is necessary in subsequent seasons.

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Press firmly using ballpoint pen. TPN orders must be initiated and renewed on this form. Write on only one column each day for 4 days; then, start new form. **Orders must be received prior to 2:00 p.m. for same day preparation.**

Date	**TODAY**			
Time	**1330**			
Patient Weight	**7.2 kg**	**kg**	**kg**	**kg**
Route of Administration	Central **X** Peripheral	Central Peripheral	Central Peripheral	Central Peripheral
LIPID 20% EMULSION				
Lipid Dose	**3** g/kg/day	g/kg/day	g/kg/day	g/kg/day
Lipid Infusion Rate (5 mL = 1 g of fat)	**9** mL/hr for **12** hr	_____ mL/hr for _____ hr	_____ mL/hr for _____ hr	_____ mL/hr for _____ hr
DEXTROSE AND AMINO ACID SOLUTION				
Infusion Volume	**612** mL/day	mL/day	mL/day	mL/day
Infusion Rate	**25.5** mL/hr	mL/hr	mL/hr	mL/hr
Dextrose Concentration	**10** %	%	%	%
Amino Acids	**3** g/kg/day	g/kg/day	g/kg/day	g/kg/day
ELECTROLYTES				
Sodium Acetate	**2** mEq/kg/day	mEq/kg/day	mEq/kg/day	mEq/kg/day
Sodium Chloride	**0** mEq/kg/day	mEq/kg/day	mEq/kg/day	mEq/kg/day
Sodium Phosphate	**0** mEq/kg/day	mEq/kg/day	mEq/kg/day	mEq/kg/day
Potassium Acetate	**2** mEq/kg/day	mEq/kg/day	mEq/kg/day	mEq/kg/day
Potassium Chloride	**2** mEq/kg/day	mEq/kg/day	mEq/kg/day	mEq/kg/day
Potassium Phosphate	**0** mEq/kg/day	mEq/kg/day	mEq/kg/day	mEq/kg/day
Magnesium Sulfate	**0.8** mEq/kg/day	mEq/kg/day	mEq/kg/day	mEq/kg/day
Calcium Gluconate	**2.5** mEq/kg/day	mEq/kg/day	mEq/kg/day	mEq/kg/day
MULTIVITAMINS AND TRACE ELEMENTS				
Multivitamins (MVI) (Use Pediatric MVI if under 11 years of age and Adult MVI if 11 or older. See reverse for MVI formulation.)	**X** Pediatric MVI (2 mL/kg up to 5 mL/day) Adult MVI (10 mL/day)	Pediatric MVI (2 mL/kg up to 5 mL/day) Adult MVI (10 mL/day)	Pediatric MVI (2 mL/kg up to 5 mL/day) Adult MVI (10 mL/day)	Pediatric MVI (2 mL/kg up to 5 mL/day) Adult MVI (10 mL/day)
Trace Elements (TE) (Use Pediatric TE if under 11 years of age and Adult TE if 11 or older. See reverse for TE formulation.)	**X** Pediatric TE (0.2 mL/kg up to 5 mL/day) Adult TE (1 mL/day)	Pediatric TE (0.2 mL/kg up to 5 mL/day) Adult TE (1 mL/day)	Pediatric TE (0.2 mL/kg up to 5 mL/day) Adult TE (1 mL/day)	Pediatric TE (0.2 mL/kg up to 5 mL/day) Adult TE (1 mL/day)
OTHER ADDITIVES				
Heparin	units/mL	units/mL	units/mL	units/mL
Physician Signature/ ID Number				
Nurse Signature				
Pharmacist Signature				

XYZ Children's Hospital
Great Healthcare System
Pediatric Total Parenteral Nutrition Form

Form Number 2300-50001 (Revised 09/2009)
White—Chart Copy All Others—Pharmacy

ADDRESSOGRAPH

Figure 1: Sample Parenteral Nutrition Form for a Pediatric Patient

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CASE 2.3
Toxicology | Level 3

Mary A. Babico and Hanna Phan

1. What subjective and objective evidence supports the diagnosis of serotonin syndrome?

SUBJECTIVE FINDINGS: The patient arrived unresponsive and has a prescription for a chronic medication (fluoxetine) that can alter serotonin concentrations in the body. An empty bottle of fluoxetine and an empty box of guaifenesin/dextromethorphan extended-release were found on site according to report.

OBJECTIVE FINDINGS: The patient has measurable ankle clonus and presented with tachycardia. Increased body temperature (39°C) also is an objective finding of serotonin syndrome. On physical exam, the patient's skin is moist (likely diaphoresis), pupils are dilated, and lower extremities are noted to be rigid and positive for Babinski signs bilaterally. Although laboratory findings are nonspecific (i.e., elevated WBC count, elevated creatinine kinase, and decreased serum bicarbonate level), these are consistent with serotonin syndrome. The urine toxicology screen was positive for amphetamine, an agent with effects that alter serum serotonin concentrations, further predisposing the patient to serotonin syndrome.

Dextromethorphan exhibits its serotonergic activity at the 5-hydroxytryptophan-2 (5-HT2) receptor along with its antitussive effects by binding to sigma opioid receptors at recommended doses. At doses exceeding approximately 1.5 mg/kg/day (or 120 mg/day in older children and adults), dextromethorphan can also impair adrenergic neurotransmitter reuptake, leading to increased presynaptic serotonin concentrations. Amphetamine derivatives cause an increase in serotonin concentrations by acting as sympathomimetic agents, stimulating the release of catecholamines from their presynaptic terminal nerve storage sites.

2. What subjective and objective evidence supports toxicities due to other ingested agents?

SUBJECTIVE FINDINGS: The patient was found unresponsive. The mother reported that an empty vial of glipizide was found in the home. Guaifenesin overdoses can also cause central nervous system (CNS) depression.

OBJECTIVE FINDINGS: The patient's blood glucose was far below normal limits along with mildly decreased serum potassium, and the patient was tachycardic. A serum acetaminophen concentration was

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detectable on lab work and dextromethorphan was found on thin layer chromatography.

An outcome of severe hypoglycemia is CNS depression due to the inadequate supply of glucose, and subsequently reduced intracellular ATP, as the brain's sole source of energy. Catecholamines, specifically epinephrine and norepinephrine, and cortisol are released in a state of hypoglycemia, which results in tachycardia. Along with cellular glucose intake, the release of insulin stimulated by sulfonylureas causes an intracellular shift of potassium resulting in low serum concentrations.

3. Devise a pharmacologic regimen for the treatment of this multiagent ingestion.

The management of serotonin syndrome involves discontinuation of all serotonergic agents and controlling precipitating symptoms. Treatment is generally aimed at eliminating neuromuscular abnormalities (e.g., muscle rigidity); controlling agitation; preventing or treating seizures; and normalizing elevations in body temperature, heart rate, and blood pressure. The administration of benzodiazepines is first-line therapy, regardless of severity, for agitation, prevention or treatment of seizures, and suppressing shivering, which can further prolong hyperthermia. Lorazepam is administered at 2 mg IV every 10 to 30 minutes as needed to control muscle rigidity, agitation, and seizure activity. Cyproheptadine, a 5-HT2A antagonist, can be utilized for additional symptom management, specifically neuromuscular abnormalities (such as muscle rigidity). Its use as an antidote has limited evidence for efficacy but is generally recommended at an initial dose of 12 mg orally, or in the case of our patient via nasogastric or orogastric tube, followed by 2 mg at 2-hour intervals until symptoms resolve. A maintenance dose of 4 to 8 mg every 6 hours has also been used until agitation improves, clonus/tremors are eliminated, and vital signs normalize along with overall clinical status. In younger children, 0.25 mg/kg/day (up to 12 mg/day) of cyproheptadine has been shown to eliminate symptoms. IV fluid replacement with boluses of 20 mL/kg isotonic solution followed by a calculated weight-based maintenance infusion can provide adequate hydration and help to prevent the acute renal dysfunction in the setting of rhabdomyolysis secondary to muscle rigidity, as indicated by patient's elevated creatinine kinase. Certain antipsychotics, specifically haloperidol and droperidol, should be avoided as these butyrophenones can both increase circulating catecholamines. This can prolong serotonin syndrome as well as inhibit diaphoresis to aid in dissipating body heat through its anticholinergic effects.

Symptomatic hypoglycemia, as seen in this patient, is generally treated with dextrose 0.5 to 1 g/kg as an IV bolus immediately. Dextrose 10% or 25% preparations are usually used in younger children and adolescents, whereas adults can be given 50%. Concentrations greater than 12.5% should be administered through a central IV line. A bolus of 500 mL dextrose 10% (D10W) can be given peripherally, or if patient has a central line, a bolus of 100 mL D25% or 50 mL D50% can be administered to this patient. Dextrose quickly raises blood glucose level and increases glucose delivery to the brain. Boluses can be repeated every 10 to 15 minutes with fingerstick blood glucose (FSBG) monitoring, and dextrose boluses may be repeated if serum glucose remains <60 mg/dL. Hypoglycemia can be prolonged and may require a continuous infusion of dextrose. Transient hyperglycemia from IV dextrose can trigger increased insulin release and recurrent episodes of hypoglycemia. To counteract this effect, octreotide, a somatostatin analog, reduces the calcium-induced release of insulin from pancreatic beta cells by inhibiting the voltage-gated calcium channel. The dosing for octreotide in the setting of sulfonylurea overdose has not been established, but studies have shown effective dosing at 1 to 1.5 mcg/kg subcutaneously every 6 to 8 hours to blunt the release of insulin until blood glucose remains stable above 70 mg/dL. Due to the long half-life of glipizide, octreotide therapy may be needed for greater than 24 hours. Glucagon is also another agent that has been used to treat hypoglycemia and can be given intravenously, intramuscularly, or subcutaneously at 0.025 to 0.1 mg/kg (maximum dose: 1 mg) every 20 minutes as needed. Glucagon has fallen out of favor due to its short duration of action as it depletes hepatic glycogen stores through

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glycogenolysis and gluconeogenesis, thus diminishing its effect over time. It may not be the preferred agent in this patient.

The time of ingestion of acetaminophen cannot be assessed with the given history, which is of importance because the antidote, N-acetylcysteine, is most effective when administered within 8 to 10 hours of ingestion. However, the serum acetaminophen concentration is not of critical clinical concern with patient's acute ingestion, and liver transaminases are within desired range; therefore, supportive care measures are indicated at this time. Based on the estimated time the patient was suspected to ingest acetaminophen, a serum drug level of 12 mcg/mL is far below the treatment line as proposed by the modified Rumack-Matthew nomogram. Signs of acetaminophen-induced hepatotoxicity can appear approximately 36 hours postingestion; thus, liver function tests should be monitored closely every 12 hours for at least 2 days to monitor increases in liver transaminases and international normalized ratio (INR), then daily thereafter until hospital discharge. Serum acetaminophen concentrations typically peak within 4 hours of drug overdose and may be prolonged in the presence of anticholinergic agents due to their effects on delaying gastric emptying. It may be reasonable to check an acetaminophen level 4 to 12 hours after the initial level to monitor for rising levels and determining treatment based on the modified Rumack-Matthew nomogram or to ensure drug clearance.

Patient's depression and management of stress warrants a psychiatry consultation, especially in light of suicidal behavior along with the recent initiation of a selective serotonin reuptake inhibitor (SSRI).

4. Assess the use of lorazepam in the treatment of this toxic ingestion.

Benzodiazepines elicit anticonvulsant and muscle-relaxing effects by binding to specific sites on neuronal gamma-aminobutyric acid (GABA) receptors resulting in enhanced inhibitory synaptic transmission. Lorazepam is the most potent of the benzodiazepines and less lipophilic; therefore, the onset of action is delayed compared to diazepam or midazolam. However, the duration of action is prolonged, which is desirable in treating and preventing seizures. Its metabolism through hepatic glucuronidation allows for less drug–drug interactions than other benzodiazepines, making lorazepam ideal in situations of multidrug ingestions. Caution should be taken when administering lorazepam to patients with hepatic dysfunction because its metabolism could be altered, as well as renal dysfunction because the IV formulation contains polyethylene glycol, which can accumulate and lead to acute tubular necrosis.

5. Which monitoring parameters should be considered for the treatment of this multiagent poisoning? How often should they be checked?

Vital signs should be monitored frequently—at least every hour initially until normalization of heart rate (normal range: 60 to 90 BPM), blood pressure (approximately 120/80), body temperature (approximately 36.5°C to 37.5°C), and respiratory rate (12 to 16 breaths per minute), especially with the use of lorazepam as it can cause respiratory depression—then in increasing intervals as patient begins to improve. Neurological tests to assess for mentation should be done every 1 to 2 hours until return to patient's baseline mental status, along with FSBG to assess the need of dextrose boluses or hyperglycemia from treatment. Once euglycemia is achieved (70 to 120 mg/dL) and confirmed with two FSBG within range approximately 30 minutes from each other, FSBG monitoring can be increased to every 2 to 6 hours. It would be reasonable to discontinue dextrose or octreotide therapy at this time; however, blood glucose should still be followed just as closely because glipizide has an extended half-life, and the patient may experience rebound hypoglycemia.

Serum electrolytes should be checked 1 to 2 times daily, or more frequently if indicated by clinical presentation, to monitor for abnormalities especially potassium as glycemic control is being established. Because the release of insulin stimulated by sulfonylureas causes an intracellular shift of potassium resulting in low serum concentrations, replacement of

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potassium should be considered if the patient becomes hypokalemic or symptomatic. The liver function panel should be monitored at least twice daily to watch for signs of acetaminophen-induced hepatitis, as discussed earlier. A physical exam should be performed every 8 to 12 hours throughout the day to monitor for symptom and clinical changes.

The common, yet infrequent, adverse reactions of cyproheptadine can present similarly to patient's altered mental status and agitation, but watching for urinary retention and nasal congestion can help in determining adverse reactions of the drug in light of serotonin syndrome. Bradycardia, diarrhea, dyspepsia, and edema are common adverse reactions of octreotide among many, less frequently occurring reactions.

6. Develop a patient education plan for this patient (and her caregiver/s) upon discharge.

The patient should take medications prescribed by her physician as ordered. Medications should not be abruptly discontinued without speaking to a healthcare professional. This is especially important with fluoxetine because mood changes and an increased risk of suicidal thoughts or behavior can been seen with sudden discontinuation. Parents/caregivers should observe the child or adolescent closely for any changes in mood and behavior throughout therapy with an antidepressant (e.g., SSRI), especially within the first few months of treatment, because studies have shown increased suicidal thoughts and behaviors in children/younger adults who take antidepressants.

The patient should not take any medications that have not been prescribed for her. The dose and indication of prescription medications are specific to the user and may not be appropriate for her or the concurrent medical issues she may have. These medications can also interact with other medications or food that can alter its effects in her body. Parents/caregivers should be advised to keep medications in a locked or safe storage area. The use of over-the-counter (OTC) medications is recommended to be supervised by a healthcare professional, and it is important to know their limits. Acetaminophen, for example, has a limit of 3 g per day, which is six extra-strength (500 mg) or nine regular strength (325 mg) tablets. Acetaminophen is also found in many combination products, such as cold and cough medications, which should also be accounted for.

STRESS MANAGEMENT: Try relaxation techniques (e.g., yoga, mediation, breathing exercises); set a schedule or create to-do lists to aid in time management; avoid areas/situations that induce stress; eat healthy; be active (e.g., exercise, volunteer, play with the dog); get a full night's sleep every night; and avoid drugs and alcohol.

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CASE 3.1
Patent Ductus Arteriosus | Level 1

Brady S. Moffett

1. What is the subjective and objective evidence for the diagnosis of PDA?

SUBJECTIVE FINDINGS: Poor feeding and lack of weight gain, sweating while feeding

OBJECTIVE FINDINGS: Premature infant, echocardiogram demonstrating a PDA, tachypnea, grade 3 murmur over left infraclavicular region, tachypnea

2. Assess the need for pharmacologic therapy for closure of a PDA.

Patients should be considered for pharmacologic closure of a PDA if they are symptomatic with signs of heart failure and pulmonary overcirculation. This patient is experiencing both of these as evidenced by poor feeding, lack of weight gain, tachypnea, and sweating while feeding. The patient is a candidate for pharmacologic therapy for closure of the PDA. If pharmacologic therapy fails, surgical intervention would be the next step.

3. Devise and assess a pharmacologic regimen for the treatment of pulmonary overcirculation and heart failure.

This patient is experiencing pulmonary overcirculation due to the shunting of blood from the systemic vasculature (aorta) to the pulmonary artery and lungs. The congestion and low cardiac output that results can be controlled by optimizing the patient's preload status. This can be accomplished through the use of diuretics and fluid restriction. Fluid restriction in the newborn child who is breastfeeding would be difficult. Loop diuretics, while useful for optimizing preload status, can also generate endogenous prostaglandin production, which can maintain the patency of a ductus arteriosus. Because the patient is symptomatic and experiencing congestion, furosemide at 1.8 mg (1 mg/kg/dose) twice daily should be initiated.

The patient should have an increase in urine output and fewer signs of pulmonary overcirculation/heart failure, which would include appropriate weight gain per age and height, lack of sweating or tiring out during feeding, and normal respiratory rate. Urine output should be monitored by diaper weight at least every 3 to 4 hours. Monitoring of serum electrolytes on a daily basis would be useful to determine the need for electrolyte supplementation, specifically sodium and potas-

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sium, which would be decreased in a patient receiving furosemide.

4. Determine the appropriate (if necessary) pharmacologic regimen for the treatment of PDA.

Many patients with a PDA will experience spontaneous closure at 48 to 72 hours of life. This patient is symptomatic at 6 days of life and therefore should receive therapy for PDA closure. After treatment for the patient's pulmonary overcirculation and congestive heart failure, pharmacotherapy for PDA closure should be initiated.

Intravenous (IV) indomethacin and IV ibuprofen (10 mg/kg/dose once, then 5 mg/kg/dose at 24 and 48 hours) have both been utilized to close a PDA. Trials have shown that they are equivalent in efficacy but that IV ibuprofen had a lower incidence of kidney injury (oliguria and rise in serum creatinine) as compared to IV indomethacin. IV ibuprofen 18 mg once, then 9 mg at 24 and 48 hours following the initial dose (10 mg/kg/dose once, then 5 mg/kg/dose at 24 and 48 hours) should be administered to close the PDA. Dosing intervals should be increased if the patient's urine output falls below 0.6 mL/kg/hr.

The patient should have a serum creatinine, chemistry panels, and complete blood counts done at baseline and daily for the duration of therapy. An echocardiogram should be performed on the day after completion of therapy to ensure that the ductus has closed.

If the ductus has not closed, a repeat course of ibuprofen can be initiated as soon as the patent ductus has been identified. Surgical intervention—whether a surgical PDA ligation or coil occlusion in the cardiac catheterization laboratory—would be indicated if the patient does not tolerate pharmacotherapy due to adverse reactions.

5. Devise pertinent education for the caregiver in regard to PDA.

Caregivers for this patient should be aware of the potential adverse reactions associated with IV ibuprofen for PDA, which include bleeding, intracranial bleeding, acute kidney injury, gastrointestinal bleeding, and bowel ischemia. The patients most at risk for these adverse reactions are premature infants (<36 weeks postmenstrual age). As this patient is older, the risk of adverse reactions is minimized.

6. Determine appropriate use of oral sucrose for pain management for minor procedures.

The patient will have multiple minor procedures performed in order to evaluate laboratory values and administer medications. These procedures potentially include heel lances and IV cannulation. Oral sucrose should be prescribed as needed for each of these procedures to minimize pain. Short-term analgesia is produced from the sucrose, and the sucking action when sucrose is placed on a pacifier and given to infant prior to and during a painful minor procedure. The analgesic effect is short lived and causes few to no adverse reactions. The dose of 24% oral sucrose solution should be 0.2 to 1 mL given on a pacifier 1 to 2 minutes prior to the painful procedure and during the procedure.

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CASE 3.2
Hypertension | Level 2

Brady S. Moffett

1. What is the subjective and objective evidence for the diagnosis of hypertension?

SUBJECTIVE FINDINGS: None

OBJECTIVE FINDINGS: Blood pressure measurements greater than the 99th percentile plus 5 mm Hg for age, gender, and height on three separate occasions (stage II hypertension); obesity (BMI 32.2 kg/m^2, 98th percentile)

RISK FACTORS: Diet (fast food and energy drinks); family history of obesity and possible type 2 diabetes mellitus (T2DM) (mother); gestational diabetes (mother)

2. What are the criteria for a diagnosis of obesity in this patient?

Obesity for a pediatric patient is based on BMI percentile for age and gender. A BMI percentile greater than 95 is considered obese. This patient's BMI is 32.2, which is greater than the 95th percentile (98.74%) for age and gender.

3. Devise a nonpharmacologic regimen for the treatment of hypertension in this patient.

The patient is currently asymptomatic. Therapeutic life modification is the primary focus for this patient (weight loss, exercise, and diet). The patient's BMI should be less than 85th percentile for age and gender, which would represent at least a ~25 kg (~62.5 lb) weight loss. Referral to nutritional counseling would be appropriate with potential referral to a physician specialist in weight loss. Exercise, including weight training and aerobic cardiovascular exercise, would be recommended after medical clearance. Limiting consumption of energy drinks and other drinks with high caffeine and sugar content should be stressed to the patient.

4. What other considerations should occur prior to initiating pharmacotherapy in this patient?

Treatment of blood pressure in children and adolescents is important, as uncontrolled blood pressure may lead to morbidity, such as hypertension and heart failure, in adult life. Assessment of blood pressure in children and obese patients can be difficult, and many factors can impact the correct assessment of hypertension. Using an appropriate

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blood pressure cuff size is critical to accurate evaluation of blood pressure in a pediatric patient, and many variations exist in manufactured blood pressure cuffs. The cuff bladder length should cover 80% to 100% of the circumference of the child's upper right arm. Patients may also experience white coat hypertension and may benefit from continuous ambulatory blood pressure monitoring.

Because this patient has had blood pressure measurements on three separate occasions and they all have been elevated, this patient meets the criteria for stage II hypertension based on height percentile, age, and gender staging criteria for hypertension. He requires pharmacotherapy to prevent end organ damage, such as left ventricular hypertrophy (LVH). A complete renal evaluation to rule out kidney abnormalities is recommended. The evaluation includes a renal ultrasound, urinalysis, serum electrolytes, serum creatinine and BUN, and urine culture. A complete blood count is also necessary to rule out anemia as a result of kidney disease.

Although the patient denies the use of illicit drugs, a urine drug screen should be obtained to rule out the presence of drugs, which could cause hypertension. The use of highly caffeinated "energy drinks" can also increase blood pressure, and the patient's blood pressure should be evaluated without their presence. Over-the-counter medications, such as pseudoephedrine, can cause elevations in heart rate and blood pressure as can prescription medications used for attention deficit hyperactivity disorder. The use of these medications should be evaluated when assessing for hypertension.

Patients with stage II hypertension should have end organ damage assessed. LVH, as consequence of uncontrolled hypertension, should be evaluated by echocardiogram. An eye exam should be performed to rule out retinal vascular changes.

Additionally, due to the patient's obesity and patient's family history of obesity and possible T2DM, a fasting blood glucose, hemoglobin A_{1C}, fasting insulin level, and fasting lipid profile should be obtained. The patient should also be asked for any signs and symptoms related to T2DM, such as increased thirst or hunger or frequent urination.

5. Devise a pharmacologic regimen for the treatment of hypertension in this patient.

The dosing strategy for medications should be to initiate the patient on the lowest recommended dose with frequent reassessment of blood pressure and upward titration of the dose as necessary. Decreases in dose of medication or discontinuation of medication may occur if etiologies for hypertension (i.e., obesity) are minimized or eliminated.

Many options are available for treatment of hypertension, and medications should be selected based on patient-specific factors. Angiotensin-converting enzyme (ACE) inhibitors are common first-line medications because many patients with hypertension also have LVH or proteinuria, but ACE inhibitors should be used with caution in patients with significant renal scarring or dysfunction. This patient does not have any signs of kidney injury based on his serum creatinine value. He should have an echocardiogram to determine the presence of LVH. Beta-blockers can be used but should be avoided in a patient using insulin for diabetes, and they may also impair athletic performance by limiting heart rate. Because this patient may have problems with blood glucose management, as evidenced by the elevated blood glucose, and is also active in sports, a beta-blocker may not be the best first-line agent for pharmacotherapy. Calcium channel blockers, specifically the dihydropyridines, have also been used. These agents can also cause hypotension and peripheral edema.

As all of the medications listed can lower blood pressure, current recommendations state that initial choice of antihypertensive therapy can be based on practitioner experience, patient preference, and response to therapy.

Enalapril at a dose of 5 mg once daily would be appropriate first-line pharmacotherapy for this patient. Reassessment of blood pressure should occur on a weekly basis, and titration upward at 2.5 mg increments would be reasonable until goal blood pressure measurements are achieved. If enalapril does not achieve blood pressure goals at a maximum dose of 40 mg per day, a second agent may be added (again, based on the practitioner's preference and experience).

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6. Devise treatment goals and outcomes for each of the identified healthcare problems presented in the patient case.

The pharmacotherapeutic goal for the patient's blood pressure is less than the 95th percentile for age, gender, and height, which would be less than 132 mm Hg systolic and less than 80 mm Hg diastolic to prevent end organ damage. Additionally, adverse events to pharmacotherapy should be minimized. ACE inhibitors may cause hypotension, which can manifest as lightheadedness or dizziness, and a dry cough can occur. Angioedema is a serious but rare adverse event associated with ACE inhibitors. The patient should be counseled on these potential adverse events and instructed to contact his healthcare provider should any of these occur.

After pharmacotherapy goals have been obtained, continued reduction of blood pressure to less than the 90th percentile for age, gender, and height is ideal and should occur by weight loss, dietary intervention, and other lifestyle modifications.

Therapeutic lifestyle modifications (including weight loss) should occur in this patient at a rate of approximately 1 to 2 pounds per week. The goal patient BMI percentile should be less than the 85th percentile for the patient's age and gender, which represents an approximate weight loss of 60 lb over the course of approximately 1 year. Patients should have a urinalysis to check for proteinuria, serum creatinine, serum potassium, at baseline and follow-up echocardiograms to evaluate LVH over the course of the next 6 months. Weekly blood pressure evaluations would be necessary until blood pressure goals are achieved. Fasting blood glucose, insulin, hemoglobin A_{1c}, and lipid profiles should also be obtained every 6 months to evaluate for hyperlipidemias and T2DM.

7. Identify and manage issues in weight-based dosing associated with obese pediatric patients.

Initial recommended dosing of enalapril in pediatric patients with hypertension is 0.08 mg/kg/dose given once daily to a maximum starting dose of 5 mg. This patient weighs 102 kg, which, when calculated by weight, would result in an initial dose of 8.2 mg per day. Correct dosing in obese patients can be difficult to determine, and it depends on the pharmacokinetic properties of the drug, such as lipid solubility and distribution into fatty tissues, and patient-specific factors, such as risk of adverse events if the dose prescribed is too high, or outcomes associated with administering an inadequate dose. In adult patients, dosing based on ideal body weight or adjusted body weight has been used but cannot be routinely recommended in pediatric patients. In this case, the patient should be initiated on maximum starting dose (5 mg once daily) and doses titrated up to goal blood pressure while assessing for adverse events associated with higher doses, such as hypotension.

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CASE 3.3
Congenital Heart Defects | Level 3

Brady S. Moffett

1. What is the subjective and objective evidence for the diagnosis of Tetralogy of Fallot?

SUBJECTIVE FINDINGS: Patient complained of chest pain and difficulty breathing, which was relieved by knee-to-chest maneuver. Patient reported instances of hypercyanotic ("Tet") spells, and lips and fingertips occasionally have turned blue.

OBJECTIVE FINDINGS: Findings include 89% oxygen saturation, echocardiogram demonstrating the pulmonary stenosis, overriding aorta, ventricular septal defect, and right ventricular hypertrophy. Patient has peri-oral cyanosis, is mildly cyanotic on exam, has a respiratory rate of 38 breaths per minute, and has a murmur.

2. Devise a pharmacologic regimen for prevention of a hypercyanotic spell secondary to Tetralogy of Fallot.

Traditional pharmacotherapy with the goal of preventing hypercyanotic spells in a patient with Tetralogy of Fallot is propranolol. It is theorized that this prevents infundibular spasm, which can block flow through the stenotic pulmonary artery and subsequently prevent right-to-left shunting of deoxygenated blood. Other beta-blockers may be used, but information is limited. The dose of propranolol in a patient with frequent hypercyanotic spells ranges from 1 to 4 mg/kg/day divided 3 to 4 times daily up to a maximum of 60 mg/day. The dose for this patient would be 35 mg orally, 3 times daily. The patient should be maintained on this regimen indefinitely or until propranolol therapy is no longer effective in preventing hypercyanotic spells and surgical intervention is indicated.

Adverse reactions to propranolol therapy include hypoglycemia, bradycardia, and reduced exercise tolerance. Heart rate should be evaluated after patients have been initiated on therapy. Patients should be educated to potential adverse reactions and report symptoms of exercise intolerance, lightheadedness, dizziness, or fainting. Some patients may experience neurologic symptoms such as vivid dreams/nightmares or cognitive dysfunction. If any of these reactions are severe enough to result in discontinuation of propranolol, other beta-blockers may be used. However, no data are available for use of other beta-blockers in prophylaxis of hypercyanotic spells in Tetralogy of Fallot.

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3. Devise a pharmacologic regimen for the treatment of a life-threatening hypercyanotic spell secondary to Tetralogy of Fallot.

Nonpharmacologic therapy includes the use of a knee-to-chest maneuver. This increases peripheral vascular resistance and left ventricular pressure preventing right-to-left shunting of deoxygenated blood. Some patients will instinctively perform this maneuver, and they are colloquially known as "squatters." Pharmacotherapy is required if nonpharmacologic measures are ineffective in stopping the hypercyanotic episode and the patient is having life-threatening symptomatology, such as deteriorations in mental status or decreased perfusion to the periphery.

Pharmacotherapy for a hypercyanotic spell should focus on the reversal of right-to-left shunting of deoxygenated blood across the ventricular septal defect. The underlying physiology for decreasing the right-to-left shunt include increasing afterload and left ventricular end systolic pressure, decreasing pulmonary vascular resistance, and relaxing (or decreasing) infundibular spasm. Treatment should be based on the severity of the hypercyanotic spell and the potential for adverse reactions associated with pharmacotherapy. Patients should be admitted to the intensive care unit if the episode is deemed life threatening.

Initially, intravenous fluids at infusion rates to reflect maintenance fluid requirements are used to improve preload and cardiac output. Hypercyanotic spells can occur in patients secondary to other illnesses, which may result in dehydration. Inhaled oxygen via face mask should be used to improve oxygenation of blood and as a pulmonary vasodilator to decrease pulmonary vascular resistance.

If these modalities do not relieve the episode, care can progress to pharmacologic therapy. Morphine (0.05 mg/kg/dose) or ketamine (0.5 mg/kg/dose) can be useful to relieve pain and anxiety and to also lower pulmonary vascular resistance. Beta-blockers (enteral or intravenous) can be used to minimize infundibular spasm, reduce afterload, and promote myocardial relaxation. Propranolol can be administered at 35 mg (1 mg/kg/dose), or, if the patient is critically ill, esmolol intravenous continuous infusion initiated at 50 mcg/kg/min can be used. Phenylephrine (0.02 mg/kg/dose) can be used in severe cases to promote peripheral vasoconstriction thus increasing left ventricular pressure and preventing right-to-left shunting of deoxygenated blood.

For the patient in this case, initiation of normal saline intravenously at 75 mL/hr and oxygen 100% via face mask at 4 L/min would be appropriate first interventions. If the patient is still in pain, a dose of 1.8 mg of morphine intravenously would be appropriate. Pharmacotherapy should escalate in intensity based on the patient status. Ultimately, patients with severe activity restrictions or life-threatening hypercyanotic spells will have to undergo surgical repair.

4. Devise pertinent patient and caregiver education for the prophylaxis and treatment of hypercyanotic spell in Tetralogy of Fallot.

Patients and caregiver should be educated on the signs and symptoms of a hypercyanotic spell and to avoid activities or situations that have precipitated these events in the past, such as vigorous physical activity, dehydration, or fever. If the patient has a hypercyanotic spell, the knee-to-chest maneuver should be performed and the patient should be taken to the nearest emergency department.

Parents and patients should be educated on the indications for propranolol therapy and the importance of compliance to therapy to prevent future hypercyanotic events. Additionally, potential adverse reactions that may be associated with propranolol therapy (hypoglycemia, hypotension, bradycardia, fatigue/decreased exercise tolerance) should be highlighted to parents and patients.

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5. Determine the most appropriate medication dosage form for propranolol in the prophylaxis of hypercyanotic spells in Tetralogy of Fallot.

Propranolol is available in tablet, extended-release capsule, and commercially available solution (4 mg/mL and 8 mg/mL). This patient would be initiated on 1 mg/kg/dose every 8 hours, which would be 35 mg orally 3 times daily. Tablet sizes are in 10, 20, 40, 60, and 80 mg sizes. Extended-release capsules are available in 60, 80, 120, and 160 mg formulations. No tablet sizes offer the appropriate dose formulation. Family and patient preferences, the ability of the patient to swallow a tablet or capsule, and the ability of a child to take medications at school should be considered prior to selecting a dosage form. The 8 mg/mL concentration would provide the most accurate initial dosage form for this patient and would result in 4.4 mL 3 times daily. The option of using a tablet or extended-release capsule should be utilized as the patient grows and gains weight.

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CASE 4.1
Asthma | Level 1

Manar O. Lashkar and Hanna Phan

1. What subjective and objective evidence exists in the patient to support a diagnosis of severe asthma exacerbation?

SUBJECTIVE FINDINGS: The patient presented with inability to breathe and poor response to albuterol inhaler. This past week, he has been requiring his albuterol use nightly, which is more frequent than usual. He has an increased requirement of albuterol in the last month, requiring two refills. He has a history of missed school days due to shortness of breath, inability to exercise, and continued coughing at night.

OBJECTIVE FINDINGS: On physical exam, SpO_2 85% on RA (<90%), RR 42 breaths per min (>40), PEF is 35% (<40% of personal best), increased anterior-posterior diameter of the chest on lung exam, and hyperinflation with nasal flaring. The patient has inspiratory and expiratory wheezing, dyspnea, and is unable to speak full sentences.

2. List risk factors that necessitate intensive care unit admission in a patient with asthma exacerbation. Is this patient considered high risk with regard to his asthma?

Patients with asthma are considered high risk for a severe asthma exacerbation, when they have (1) previous severe exacerbation (e.g., ICU admission); (2) two or more hospitalizations or more than three ED visits in the past year; (3) use of greater than two canisters of albuterol in a month; (4) difficulty perceiving airway obstruction or the severity of asthma worsening; (5) low socioeconomic status or inner-city residence; (6) illicit drug use; (7) major psychosocial problems or psychiatric disease; or (8) comorbidities such as cardiovascular disease or other lung disease. High-risk asthma patients require additional care components including intensive asthma education, monitoring, and monthly follow-up.

This patient would be considered high risk for a severe asthma exacerbation given the following factors specific to his case: (1) previous severe exacerbation (i.e., previous ICU admission); (2) two hospitalizations in the last year; (3) difficulty perceiving airway obstruction or the severity of asthma worsening (the patient has always had breathing problems and did not follow up with his pediatrician); and (4) major psychosocial problems or psychiatric disease given his diagnosis of ADHD.

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3. Determine the classification of asthma control for the patient. Which components of control did the patient present with to support this classification?

The patient presented with cough throughout the day with nighttime awakenings more than twice a week. His asthma is interfering with his normal activity to the point where routine activities like gym class or even attending school is a challenge. He requires the use of his rescue inhaler several times a day and throughout the night. His lung function is 35% (less than 60% of personal best using PEF). Given this level of impairment of normal activity and need for rescue therapy, his asthma is currently considered "very poorly controlled" and will require step up (1 to 2 steps) of his chronic asthma regimen once his acute exacerbation is effectively treated. He should be re-evaluated in clinic 2 weeks after discharge and followed up regularly (e.g., every 4 weeks) until his asthma is under better control.

4. Assess the patient's current acute pharmacologic therapy for asthma.

The goals of therapy in a patient with acute asthma exacerbation are to establish control of the asthma (i.e., reduce symptoms, maintain oxygenation, eat and drink without difficulty, and prevent adverse drug reactions). The patient presented with a severe asthma exacerbation, based on subjective and objective findings. The patient was started on a systemic corticosteroid in the ED, but the dose was likely not absorbed due to emesis soon after oral dose of prednisolone. The patient will need to be started on intravenous corticosteroid until he can tolerate oral intake. The recommended corticosteroid dose is methylprednisolone at 2 mg/kg/day divided q 12 hr (methylprednisolone 30 mg IV q 12 hr). Systemic steroid dose may be reduced to 1 mg/kg/day and changed to oral route (e.g., prednisone 30 mg tablet po daily) once patient is on less frequent albuterol nebulization (e.g., q 2 hr dosing) and able to tolerate oral intake as a result of improved respiratory symptoms (reduced tachypnea and dyspnea). Current use of continuous albuterol is appropriate and patient's response to therapy should be monitored (e.g., reduced shortness of breath and wheezing) as well as adverse reactions such as tachycardia and hypokalemia. Albuterol dose and frequency may be reduced as patient's symptoms improve (respiratory rate, oxygen saturation, accessory muscle use, air exchange, wheezing, and inspiratory to expiratory ratio). These parameters should be monitored every 2 hours initially until the albuterol frequency is reduced to every 2 hours. When this is achieved, monitoring would continue to be every 2 hours until the frequency of albuterol can be decreased to every 4 hours. After that, the patient can be monitored every 4 hours, and if his symptoms are stable and did not require any other intervention, he can be discharged.

5. Identify a potential medication-related adverse reaction associated with asthma exacerbation therapy and its management.

The patient is hypokalemic (potassium of 3 mmol/L) secondary to continuous albuterol infusion. Beta-2 agonists can shift potassium into the cells leading to hypokalemia. The addition of potassium chloride to IV fluids (D5W–½NS with 20 mEq KCL per liter) is recommended. This effect will be diminished as albuterol frequency is decreased and patient starts on oral diet.

6. Devise a discharge pharmacologic regimen for his asthma and other conditions.

The desired outcomes for this patient include reduction of both impairment and risk. Reduction of impairment includes a decrease in the use of his rescue inhaler to less than two times per week, limited interference in normal activity, nighttime symptoms less than two times per month, and lung function as measured by greater than 80% of his personal best PEF. Reduction in risk includes decrease in exacerbations requiring oral corticosteroids to less than once a year as well as prevention of treatment-related adverse reactions.

Given this acute exacerbation resulted in admission to the PICU, increasing chronic therapy by 2 steps may be warranted. Based on the patient's age and current chronic asthma regimen, he is on step 2 asthma therapy with low-dose inhaled corticosteroid (fluticasone

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44 mcg 2 puffs bid). To continue his systemic corticosteroid regimen, recommend prednisone 30 mg tablet po daily to complete a total 5-day regimen of systemic corticosteroid (including both IV and po therapy) without the need to taper down the therapy because this short course (less than 2 weeks of systemic therapy) is unlikely to induce adrenal access suppression. Given the severity of his persistent asthma and classification of severity as "very poorly controlled," recommend changing his therapy to medium dose inhaled corticosteroid (fluticasone 110 mcg 2 puffs bid with spacer) plus montelukast 5 mg po daily (preferably at night). The addition of montelukast, a leukotriene receptor antagonist, may also help in managing allergy-related triggers for his asthma. Given his limitations in gym class due to his asthma, a dose of albuterol (i.e., 2 puffs) prior to exercise may be helpful. Recommend continuation of the loratadine 5 mg po daily as well as his ADHD medication, methylphenidate LA 30 mg po daily.

Patients with asthma are particularly at risk for complications of influenza infection. Thus, this patient needs to update his vaccinations by obtaining an inactivated influenza vaccine (IIV) intramuscularly prior to discharge and then annually. Intranasal live-attenuated influenza vaccine is not an option for asthma patients due to the risk of developing wheezing and an asthma exacerbation. If the patient has never received an influenza vaccine previously, he will need a total of two doses of IIV separated by at least 1 month this year only (because the patient is younger than 9 years of age) and then one dose annually.

Patient and caregiver should be educated about the possible adverse reactions of prednisone, including changes in mood and insomnia. Recommend to the caregiver to administer the dose in the morning with food to avoid an upset stomach. Also, inhaled corticosteroids have the risk of developing oral thrush if the patient does not rinse his mouth after each use.

Patient and caregiver should be educated on the use of his inhalers, including use of a spacer and the difference in roles between controller and rescue inhaler. This is particularly important to help in improving medication adherence of this patient. Also, education regarding asthma triggers (e.g., respiratory infection/illness, allergens, irritants) should be a part of the patient counseling session. An asthma action plan should be developed for the patient (see **Figure 1**) with one copy for home, one copy for school, and any additional copies for any other care providers for patient (e.g., grandmother's house afterschool).

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PATIENT NAME: PATIENT **DOCTOR**: DR. SMITH **DATE**: 3/14
DOCTOR CONTACT NUMBER: 555-555-1234
EMERGENCY DEPARTMENT NUMBER: 555-555-5678

GREEN ZONE • No cough, wheeze, chest tightness, shortness of breath during day or night • Able to do usual activities Peak flow: >80% of personal best = >160	**Take these medicines every day for asthma control:** • Fluticasone HFA MDI 110 mcg inhaler, 2 puffs with spacer inhaled twice a day • Montelukast 5 mg tablet by mouth every night **Before exercise (e.g., gym class):** • Albuterol HFA MDI inhaler, 2 puffs with spacer, 5 min before exercise

YELLOW ZONE
• Cough, wheeze, chest tightness, shortness of breath
• Waking up at night because of asthma
• Not able to do some usual activities
 	
Peak flow: Between 60% to 80% of personal best = 120–160
	1st:
	• Add quick-relief or rescue inhaler—KEEP TAKING YOUR GREEN ZONE MEDICINES
	  ○ Albuterol HFA MDI inhaler, 2 puffs every 20 min as needed
	
	2nd:
	• If your symptoms return to GREEN ZONE after 1 hr of the above treatment,
	  ○ Continue to monitor to make sure you stay in the GREEN ZONE
	  ○ Continue your GREEN ZONE medications as prescribed
	  ○ Use albuterol HFA MDI 2 puffs every 4 hr as needed
	
	• If your symptoms get worse within the hour or your symptoms do NOT return to GREEN ZONE after 24 hr,
	  ○ Take albuterol MDI HFA inhaler 4 puffs every 20 min
	  ○ Contact your doctor or go to the ED

RED ZONE
• Very short of breath, quick-relief/rescue medications have not helped
• Symptoms are the same or get worse after 24 hr of YELLOW ZONE treatment
• Cannot do usual activities
 	
Peak flow: <60% of personal best = <120
	Take this medicine:
	• Albuterol HFA MDI inhaler, 4 puffs
	• Call your doctor NOW and go to the hospital, or call an ambulance if the following occurs:
	  ○ You are still in RED ZONE after 15 min AND
	  ○ You have not reached your doctor

FIGURE 1. Example Asthma Action Plan

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CASE 4.2
Cystic Fibrosis | Level 2

Hanna Phan

1. What is the subjective and objective evidence for the diagnosis of CF acute pulmonary exacerbation in this patient?

SUBJECTIVE FINDINGS: One-week history of increased cough, increased sputum production; sick contacts at home; increased work of breathing, respiratory distress; patient more tired in the last few days, missed school days due to illness, decreased appetite

OBJECTIVE FINDINGS: Significant decline from baseline FEV_1% predicted (from 98 to 82); increased respiratory rate (24 breaths per minute), crackles and rhonchi on RLL and LUL; increased WBC; clubbing of upper extremities indicating chronic poor oxygenation, reduced oxygen saturation (SpO_2 87% on room air)

Sputum culture results, although used in guiding antimicrobial therapy selection, are not indicative of CF acute pulmonary exacerbation. Patients with CF are often colonized with pathogens in their airways. Thus, presence of pathogens as part of routine workup of patients with CF (e.g., during quarterly clinic visit) does not determine presence of CF acute pulmonary exacerbation. Patient presentation (as noted in the above subjective and objective findings) and changes in spirometry (when applicable, infants and young children are not capable of coordination for standard spirometry) are most commonly used to determine whether a patient is presenting with CF acute pulmonary exacerbation.

2. Describe recommended nonpharmacologic therapy as part of treatment for this patient's CF acute pulmonary exacerbation.

Airway clearance is an important aspect in the maintenance of lung health in patients with CF. This patient, like most of those diagnosed with CF, produces thick mucus, which is difficult to clear due to cilia dysfunction and increased viscosity. The airway obstruction, mucous plugging, and consequential airway inflammation warrant the use of physiotherapy (i.e., airway clearance) to help loosen the mucus to expel it from the airways. This patient uses a "vest," which is a high-frequency chest wall oscillation (HFWO) vest. This device works by generating airflow velocities that lead to shear forces similar to a strong cough, decreasing mucous viscosity. Airway clearance helps to move secretions, including mucus, to the larger airways so that a patient may cough the mucus up (expel). Patients with CF require complete airway clearance therapy daily as part of chronic treatment, with a recommended frequency of twice daily. With an acute

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CF pulmonary exacerbation, the frequency of airway clearance should be increased to 3 or 4 times daily at home to help expel the increased volume of sputum production during the course of an exacerbation. In the case of admission for treatment of an exacerbation, airway clearance should be done 4 times daily, because there is less of a scheduling issue with availability of the patient for more frequent physiotherapy during a hospital admission.

3. Develop a pharmacologic regimen for the treatment of CF acute pulmonary exacerbation for this patient, including goals of therapy and monitoring parameters.

The overall treatment goals of CF acute pulmonary exacerbation in CF are to recover the acute decline of lung function (i.e., FEV_1) with return to baseline lung function, reduce pulmonary symptoms including frequency of cough and sputum production, while also minimizing adverse drug reactions.

Antimicrobial Therapy

The overall goal of this patient's antimicrobial therapy is not necessarily the eradication of bacteria; however, treatment with systemic antimicrobials help in decreasing bacterial burden and growth as these may be increased during CF acute pulmonary exacerbation. Different from systemic antimicrobial therapy (e.g., IV or oral antibiotics), the use of inhaled antimicrobials (i.e., Tobi Podhaler®) as part of chronic therapy (i.e., every 28 days on and off) is to chronically suppress bacterial growth in the airways. The standard of care for treatment of a CF acute pulmonary exacerbation includes the use of systemic antimicrobials (if suspect bacterial etiology) in conjunction with increased frequency of airway clearance.

Antimicrobial therapy should be targeted against MRSA and *P. aeruginosa* based on his recent sputum culture and history of these pathogens. Recommended therapy includes two agents (from different classes) for *Pseudomonas* coverage and single drug coverage for MRSA, based on guidelines and standard of care in CF centers. The choice of antibiotics should be based on various factors, including patient-specific pathogen susceptibility data, patient medication allergies or intolerances, organ function (renal and hepatic), and concurrent drug therapy. This patient is allergic to sulfa agents with a reaction of hives and also has a noted allergy to penicillin; however, the patient is able to tolerate cephalosporins.

Larger overall doses for antimicrobials are warranted in patients with CF due to the need for higher serum concentrations for optimal pulmonary tissue penetration and increased clearance of antimicrobials noted in this population. Based on this patient's sputum culture susceptibilities and known allergies, an appropriate therapy selection for *Pseudomonas* coverage would be cefepime (50 mg/kg/dose IV q 8 hr; 1,300 mg IV q 8 hr) plus once daily dosing tobramycin (10 mg/kg/dose IV q 24 hr; 260 mg IV q 24 hr). For MRSA coverage starting vancomycin (15 mg/kg/dose IV q 6 hr; 400 mg IV q 6 hr) would be appropriate based on culture data and patient allergies. Linezolid should be reserved for cases of poor clinical response (i.e., failure to improve in respiratory symptoms after at least 3 days of therapy or improved lung function on spirometry after 1 week). A tetracycline, such as doxycycline or minocycline, could be considered second line because there are greater data supporting use of vancomycin in patients with CF acute pulmonary exacerbations. Duration of therapy is typically 10 to 14 days and will vary depending on clinical response (e.g., respiratory symptoms such as cough and sputum production, spirometry).

General clinical response should be assessed daily and includes assessment of frequency of cough, sputum production, respiratory rate, oxygen saturation, and need for oxygen supplementation. In addition, spirometry/pulmonary function tests to evaluate lung function (e.g., FEV_1) can be used to assess efficacy of treatment, to be done approximately each week. Therapeutic drug monitoring is necessary and includes tobramycin serum concentrations to be drawn after the second dose of tobramycin at two postdose times, such as 2 and 8 hours postdose. These serum concentrations are used to calculate peak and trough, with goal peak of 20 to 30 mcg/mL and trough of less than 1 mcg/mL, with C_{peak} calculated to 30 minutes after end of

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infusion and C_{trough} calculated to immediately prior to the time of the next scheduled dose. Although first-dose kinetics are possible with once-daily extended interval aminoglycosides, patients with CF acute pulmonary exacerbation can present dehydrated on admission. Thus, serum concentrations should be drawn after the second dose to allow for a day of rehydration with maintenance fluids. For vancomycin, the goal trough serum concentration is 15 to 20 mcg/mL and should be drawn prior to the fourth dose, the commonly assumed timing to achieve steady-state plasma concentrations.

In addition to monitoring for clinical improvement, the patient should be monitored for changes in renal and hepatic function while on antibiotics (e.g., BUN, serum creatinine, LFTs) at least weekly. Urine output should be assessed daily because this also reflects changes in renal function. Fluid status changes should be monitored (e.g., signs of edema) daily as this can impact pharmacokinetics of antimicrobials such as aminoglycosides.

Other Pulmonary Drug Therapy

The overall goal of other pulmonary drug therapy (i.e., albuterol, dornase alfa) is to help facilitate sputum and mucous expectoration in conjunction with chest physiotherapy, thereby helping clear airways for improved respiratory function (i.e., oxygen saturation, respiratory rate, cough, and sputum production).

Administration order of inhaled pulmonary therapy is important as each drug contributes action in airway clearance that is optimized when used in specific order. First, albuterol opens the airways through its bronchodilator action and also reduces bronchospasm from other inhaled medications such as dornase alfa, hypertonic saline, and antibiotics. The frequency of albuterol dosing is increased to pair with chest physiotherapy sessions. Thus, frequency of albuterol should be 4 times daily, paired with airway clearance. Following albuterol, either dornase alfa or hypertonic saline is administered. Dornase alfa is a mucolytic agent, cleaving DNA from degenerating neutrophils that compose the mucus in airways of patients with CF. Hypertonic saline helps thin mucus by increasing volume of the epithelial lining of the airway, thereby facilitating ciliary flow and sputum expectoration. Similarly, dornase alfa and hypertonic saline may be dosed more frequently to twice a day, with each drug regimen to pair with two of the four chest physiotherapy sessions per day. If the patient presented with significant hemoptysis (e.g., teaspoons of blood), inhaled therapies and airway clearance may be temporarily held to prevent worsening hemoptysis.

4. Evaluate the current regimen for chronic CF in this patient and recommend any necessary changes.

Pulmonary Drug Therapy

Goals for chronic pulmonary therapies in CF include (1) preserve lung function or decrease rate of decline of FEV_1; (2) complement airway clearance (i.e., chest physiotherapy) by aiding expectoration of mucus and sputum; and (3) minimize frequency of acute exacerbations.

Patient is currently in his "on" cycle for his Tobi Podhaler® for *P. aeruginosa* chronic suppression. Given the use of systemic, IV tobramycin as part of the antimicrobial regimen for this patient's CF acute pulmonary exacerbation, his Tobi Podhaler® should be held because concurrent use of inhaled tobramycin (although a small amount) may impact serum concentrations and therapeutic drug monitoring. Also dual use of inhaled and systemic therapy may increase risk for adverse reactions including tinnitus.

The patient is also on fluticasone and cetirizine for his allergic rhinitis. The patient should continue these agents during admission; there is no conflict in continuing these agents during treatment for his CF acute pulmonary exacerbation. A nasal polyp was noted on exam while in clinic. Given the patient's history of allergic rhinitis and this finding, it may warrant starting a nasal steroid such as mometasone (one spray per nostril daily).

Azithromycin in this patient is used for its anti-inflammatory properties, not as an antimicrobial therapy in CF. He should continue his azithromycin therapy during admission; there is no conflict in continuing this agent.

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Also an anti-inflammatory agent, high-dose ibuprofen has been shown to decrease the rate of decline in FEV_1 in pediatric patients. Given the nephrotoxic nature of high-dose ibuprofen and systemic tobramycin, it is recommended that the ibuprofen therapy be held during his admission and resume once the systemic tobramycin course is complete. After treatment course for his CF acute pulmonary exacerbation is complete, his ibuprofen regimen may be restarted based on evaluation of his ibuprofen serum concentrations provided. Goal ibuprofen serum concentrations for CF are 50 to 100 mcg/mL. Assuming the serum concentrations were drawn accurately for timing (i.e., drawn at 1-, 2-, and 3-hours postdose at steady-state, serum concentrations), the dose should be increased (e.g., 600 mg po bid) once it is restarted due to subtherapeutic concentrations. Ibuprofen serum concentrations should be redrawn after modifying the dose after at least 7 to 10 days of consistent dosing.

Nutrition/GI Drug Therapy

Goals for nutrition/GI drug therapy in CF include (1) encourage growth (especially in childhood); (2) maintain goal body mass index (i.e., growth overall in height and weight); (3) prevent nutritional deficiencies (e.g., fat-soluble vitamin deficiency) and associated manifestations; and (4) minimize complications of GI comorbidities (e.g., GERD, distal intestine obstruction syndrome).

The patient's pancreatic enzyme dosing is near maximum at 9,692 units lipase/kg/day (max 2,500 units lipase/kg/meal, 10,000 units lipase/kg/day) if one assumes three meals and three snacks a day. Despite high doses of enzymes, the patient is having difficulty gaining and sustaining weight. The patient has a reduced appetite, noted to have started in the last month. Based on a diet history from this clinic visit, the patient is currently on a high-calorie diet and supplements with BOOST Kids®. Further investigation about the patient's diet at home may help provide additional insight as to why he is having difficulty gaining weight. Other studies such as a metabolic cart to determine the patient's estimated kcal needs per day may be helpful as part of additional evaluation given his increased metabolism secondary to CF.

The patient is on lansoprazole for treatment of GERD. He should continue his lansoprazole therapy during his admission because there are no contraindications with its use during his CF acute pulmonary exacerbation treatment. Because efficacy of pancreatic enzyme therapy can vary based on GI tract pH, the use of a proton pump inhibitor such as lansoprazole may also optimize efficacy of pancreatic enzyme therapy when enzyme dose is maximized.

Patients with CF also often present with pancreatic insufficiency, resulting in malabsorption of fat-soluble nutrients, including fat-soluble vitamins (i.e., A, D, E, K). This patient is on AquADEK®, a type of multivitamin that has a greater amount of vitamins A, D, E, and K. These types of supplements, for patients with CF, come as a micelle-like formulation for improved bioavailability for patients with pancreatic insufficiency. The dose of this particular product for this patient's age (i.e., age 9 years and older) should be twice daily. In addition, his vitamin serum concentrations (A (retinol) = 0.15 mg/L, E (alpha tocopherol) = 4.5 mg/L, and D 25-OH = 20 ng/L) are subtherapeutic further necessitating the need to increase his dose of AquADEK®. His vitamin D serum concentration is low at 18 ng/mL, even with the increase in AquADEK® dose, this would not likely provide sufficient vitamin D to increase his serum concentration to a goal of 30 mg/mL. This patient should be started on a supplemental vitamin D_3 regimen of 5,000 units po daily. A vitamin D 25-OH total serum concentration should be obtained in 3 months, as it will take this period of time to achieve appropriate steady state serum concentration.

After the patient's CF acute pulmonary exacerbation is resolved, the patient should be scheduled for an oral glucose tolerance test to screen for CF-related diabetes (CFRD). Starting at 10 years of age, patients with CF should undergo annual screening as the incidence of CFRD increases. He should not be screened during an acute illness as the exacerbation may affect his serum glucose concentrations thereby resulting in false, unreliable results to interpret for the diagnosis of CFRD.

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5. Identify other medication-related problem(s) and develop a solution.

Until recently, the patient's family declined childhood routine immunizations. As per the patient immunization history, the patient has not received his influenza vaccine. This should be administered prior to discharge.

The patient has noted decreased appetite for the last month. It should be noted that the patient was started on methylphenidate LA 40 mg po daily a month ago for his recent ADHD diagnosis. Stimulants such as methylphenidate can decrease appetite and thus may have contributed to this patient's limited caloric intake and subsequent weight loss. A dose of 40 mg is high for a methylphenidate naïve patient. It would be appropriate to reduce his dose to 20 mg po daily, the lowest possible dose, and monitor for efficacy and increase dose, if needed, at weekly intervals by 10 mg/day. The dose reduction may improve the patient's appetite and weight.

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CASE 4.3
Respiratory Distress Syndrome and Bronchopulmonary Dysplasia | Level 3

Hanna Phan

1. What is the subjective and objective evidence for the diagnosis of BPD in this patient?

SUBJECTIVE FINDINGS: Increased work of breathing, tachypnea, retractions, pulmonary decompensation despite use of albuterol, edema, and difficulty weaning ventilator settings, including supplemental oxygen

OBJECTIVE FINDINGS: History of RDS requiring treatment with surfactant, chest radiograph showing diffuse haziness throughout, hypoinflation on RLL, rule out cardiac etiology given ECHO is normal, inability to wean from ventilator, respiratory rate (35 breaths per minute, above ventilator set rate in SIMV mode); on physical exam, course rhonchi noted, diffuse rales and wheeze on auscultation, edematous in appearance

2. Describe the risk factors for, clinical presentation of, and recommended treatment for neonatal RDS.

Risk for RDS is dependent on gestational age and birth weight as necessary amount of surfactant for normal lung function occurs at approximately 36 weeks gestation. As such, the risk for RDS is increased for neonates born less than 36 weeks gestation, but it is highest for those born less than 30 weeks gestation. Surfactant serves to lower surface tension in the alveoli, thereby helping to keep them open for air exchange and prevention of alveolar collapse. Surfactant is crucial at around 36 weeks gestational age in the saccular phase of lung development. Inadequate surfactant can result in atelectasis and impaired gas exchange and ultimately RDS. The administration of antenatal corticosteroids encourages the maturation of surfactant production in the fetus. This patient was born at an extremely low birth weight of 800 g at 26 weeks gestation, and his mother did not receive antenatal corticosteroids, which placed him at risk for RDS.

Neonates with RDS present with respiratory distress, including tachypnea, intercostal retractions, grunting, and cyanosis. The onset of symptoms is usually soon after delivery (e.g., a few hours after birth). Laboratory findings may include hypoxemia, hypercapnia, and mixed acidosis on blood gas measurements. The neonate will also likely have increased oxygen requirements and may require mechanical ventilation to maintain respiratory function. On chest radiograph, diffuse reticular-granular opacification with defined large airways may be noted. Given the overlap of signs and symptoms with other conditions,

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such as neonatal sepsis, a differential diagnosis is done by considering the onset of symptoms, clinical presentation, and objective findings such as the chest radiograph.

Given the etiology of RDS is centered on the lack of surfactant, recommended treatment for RDS is the administration of exogenous surfactant via the endotracheal tube. The administration of surfactant replaces the deficiency due to prematurity. Clinical trials have demonstrated the efficacy of surfactant resulting in reduced risk of acute pulmonary injury (pneumothorax, interstitial emphysema), development of chronic lung disease, and mortality. Early administration (within 2 hours of birth) has been shown to improve these outcomes versus delayed administration. Patients who may benefit most from surfactant therapy with regard to mortality are those born less than 30 weeks gestational age or less than 1,250 g. Surfactant is available as both synthetic and natural in origin. Administration of surfactant can be considered prophylactic in nature, which is given within 30 minutes of birth, and rescue, which is administered once the diagnosis of RDS is established. Early rescue is defined as administration within 2 hours of birth; delayed rescue is after 2 hours following birth. One dose is given initially and then, depending on the specific surfactant, doses may be repeated up to four total doses. Patients who require mechanical ventilation for respiratory support with oxygen requirements exceeding FiO_2 30% may benefit from repeat dosing. Administration of repeat doses is dictated by symptoms (e.g., tachypnea, retractions) persisting during the observation period after the first dose, which is typically the dosing interval of the specific agent. The number of repeated doses is dependent on selected agent, ranging from two to four doses.

Beractant (4 mL/kg/dose) and calfactant (3 mL/kg/dose) are bovine derived, both with potential for up to four repeated doses, at 6 and 12 hours between doses, respectively. The newly approved synthetic surfactant, lucinactant, dosed at 5.8 mL/kg/dose for up to three doses separated by at least 6 hours, contains a peptide to mimic human surfactant protein B and has no animal-derived components. This patient received poractant alfa, which is porcine derived, dosed at 2.5 mL/kg initially followed by 1.25 mL/kg for two subsequent doses at birth, doses separated by 12 hours. Selection of surfactant product is dependent on institution formulary (i.e., usually institutions select one agent), which is based on available, limited comparative efficacy data and cost. Patients receiving surfactant should be continuously monitored for lung volume and compliance as evidenced by persistence of symptoms of respiratory distress (tachypnea, poor oxygenation, increased work of breathing), and requirement for respiratory support (e.g., mechanical ventilation).

3. Discuss approaches in the prevention of BPD in premature neonates.

BPD is a long-term complication of RDS and is also called *chronic lung disease of infancy*. BPD can arise from any acute lung disorder that results in respiratory failure and need for long-term mechanical ventilation (e.g., a month) for respiratory support. Clinicians now view BPD differently due to advances in perinatal care and neonatal respiratory therapy. The "new BPD" is now clinically milder and is characterized by reduced alveolar development secondary to prematurity, still involving airway injury with inflammation and fibrosis. BPD was more severe in nature due to lung injury from older mechanical ventilation methods, which resulted in more aggressive therapy. Additionally, the manifestation of disease was longer in duration. Instead of being considered a disorder primarily due to lung injury due to respiratory support from mechanical ventilation, BPD is a developmental disorder ultimately due to prematurity. Aside from preventing BPD through prevention of RDS by the administration of surfactant, other pharmacotherapy has been studied.

Vitamin A is an antioxidant needed for tissue differentiation and cell growth. Premature neonates, especially those born with extremely low birth weight, are deficient in vitamin A. Studies have shown reductions in BPD and mortality in premature neonates with a birth weight less than 1,000 g, when vitamin A 5,000 units was administered 3 times a week for 4 weeks. This patient received vitamin A as a measure to prevent BPD development.

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Inhaled nitrous oxide has also been proposed to prevent BPD in premature neonates requiring mechanical ventilation due to respiratory failure when birth weight exceeds 1,000 g. However, due to limited evidence and cost, it is not routinely recommended for BPD prevention. Caffeine citrate (loading dose of 20 mg/kg followed by maintenance dose of 5 mg/kg until apnea resolves), a respiratory stimulant often used for treatment of apnea of prematurity, may reduce the need for positive pressure ventilation and oxygen supplementation, thereby reducing risk of developing BPD. In cases of neonates with severe apnea, the use of caffeine may prevent development of BPD through indirect means by reducing duration of positive pressure ventilation and supplemental oxygen. Thus, the use of caffeine citrate as a preventive therapy should be considered in premature infants who are at risk of severe apnea.

4. Develop a pharmacological regimen for the treatment of BPD for this patient. Why are some previously used medications, such as systemic corticosteroids, no longer recommended?

The overall goal of treatment for BPD is to improve clinical symptoms, including work of breathing, respiratory rate, and oxygen saturation, thereby facilitating mechanical ventilator and oxygen support weaning and preventing further lung injury.

First, preventing lung injury due to mechanical ventilation and supplemental oxygen is essential in reducing the development or worsening of BPD severity. This patient is currently mechanically ventilated with a high oxygen requirement of FiO_2 at 50%. Weaning the ventilator settings, including oxygen support, to as close to room air as possible will help prevent further lung injury due to the pressure from mechanical ventilation and oxygen radicals from a high FiO_2. The patient is edematous, which likely includes pulmonary edema that can compromise lung compliance making it difficult to wean ventilator settings. Thus, the patient should be started on fluid restriction, less than 150 mL/kg/day, which is 225 mL/day in this patient. Fluid restriction is challenging with current nutritional needs required for optimal growth and sufficient urine output.

The use of diuretics can help alleviate pulmonary edema, thereby improving pulmonary function and allow for weaning of ventilator support. The choice of diuretic can depend on the acuity of the BPD; in this case, the patient is unable to be weaned from the ventilator, requires high oxygen supplementation, and is edematous. His BPD is acute in nature at this time necessitating the use of a loop diuretic, such as furosemide (e.g., 1 mg/kg/dose or 1.5 mg po/IV bid). Monitoring while on furosemide would include monitoring of renal function (BUN, serum creatinine) and electrolytes every other day during acute treatment. Chronic use of furosemide has potential adverse reactions including electrolyte disturbances (reduced serum K+, Cl-, Na+), ototoxicity, osteopenia, and nephrocalcinosis/nephrolithiasis. Given its adverse reactions in the short- and long-term, furosemide should be used sparingly and as short of duration as possible. For chronic diuretic management, thiazide diuretics, such as chlorothiazide, can have reduced effect on electrolytes and thus is a preferred diuretic for chronic treatment of BPD, especially those who are ventilator-dependent such as this patient. For this patient, chlorothiazide 20 mg/kg/day divided bid (15 mg po bid) would be appropriate to help improve pulmonary function. The dose can be increased every 3 to 7 days, up to 40 mg/kg/day divided bid based on clinical response (e.g., work of breathing, ability to wean ventilator settings and oxygen supplementation). Electrolytes should be monitored every 3 days for the first 2 weeks of therapy and then at least weekly thereafter with chronic thiazide use. Supplementation of electrolytes (e.g., potassium or sodium) may be necessary when using higher doses of loop or thiazide diuretics. Urine output and changes in respiratory function (i.e., ability to wean ventilator settings, oxygen requirement) should also be monitored routinely (e.g., daily) while the patient is on diuretics.

Albuterol should be continued as bronchodilators aid in reducing airway hyperactivity and airway resistance, and improving lung compliance. In combination with fluid restriction

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and diuretics, albuterol serves as a potentially useful adjunctive therapy for this patient's acute BPD. Given the limited response to albuterol at this time, it may be appropriate to consider the addition of ipratropium (e.g., 250 mcg nebulized 4 times daily), an anticholinergic, to help improve symptoms and help reduce airway obstruction. Improvement in symptoms (work of breathing, respiratory rate, ability to wean ventilator settings) should be monitored daily.

Older data on systemic corticosteroids, such as dexamethasone, demonstrated efficacy as an anti-inflammatory agent for BPD. Corticosteroids, both systemic and inhaled, help suppress neutrophil migration and production of inflammatory mediators, demonstrating some beneficial role in BPD. However, systemic corticosteroids, such as dexamethasone, have fallen out of favor due to its long-term sequelae such as growth failure, hyperglycemia, hypertension, cardiomyopathy, and impact on neurodevelopment. This patient was started on dexamethasone at BPD dosing based on previous studies (0.5 mg/kg/day divided bid for 3 days, then 0.25 mg/kg/day divided bid for 3 days, then 0.12 mg/kg/day divided bid for 3 days, then 0.05 mg/kg/day divided bid for 3 days); however, given the risks outweighing benefits and lack of a prior trial on other, less harmful interventions (e.g., fluid restriction, diuretics), it should be discontinued at this time. Patient has only received 1 day of treatment and thus adrenal suppression is less likely. Inhaled corticosteroids, such as fluticasone, have been proposed for use in treatment of BPD; however, there is limited efficacy and safety data available to support routine use in BPD. Use of inhaled corticosteroids is considered when other therapies provide limited response (i.e., inability to wean ventilator settings, decrease oxygen supplementation). If the patient continues to have respiratory distress following aforementioned interventions, a trial of inhaled corticosteroids may be warranted.

5. Identify the medication-related problem(s) and develop a solution.

The use of dexamethasone for treatment of BPD is inappropriate as described.

The patient had increased respiratory secretions, which is seen in some mechanically ventilated patients, and was started on glycopyrrolate for treatment. The patient's dose of glycopyrrolate for his weight was started at the maximum dose (100 mcg/kg/dose, 150 mcg/dose, or 0.15 mg/dose for this patient), and the patient is experiencing reduced urine output. The addition of the diuretics for BPD and a dose reduction of the glycopyrrolate may help improve urine output. Glycopyrrolate should be used at lower doses (40 mcg/kg/dose, 60 mcg/dose, or 0.06 mg for this patient) for a few days and tapered to a lowest effective dose if possible.

6. When is a patient with BPD considered ready for discharge? Describe what should be included in caregiver education for discharge.

This patient will need to remain in the NICU for growth given his prematurity. However, criteria to consider possible discharge home would include (1) ability to sustain temperature regulation (i.e., does not require a heated incubator); (2) ability to sustain appropriate weight and demonstrate normal growth; and (3) respiratory stability (no distress, without significant oxygen supplementation need) with regard to respiratory status during sleep, feedings, and activity. If he requires oxygen supplementation at home, this is often paired with periodic pulse oximetry monitoring at home. His caregiver would need to be educated as to when to seek medical attention, including times at which there are increased symptoms of respiratory distress and decompensation. His caregiver would also need to be trained on cardiopulmonary resuscitation (CPR), equipment use if the patient is going home on oxygen monitoring, and nutrition requirements. Medication education would include proper measurement of doses, storage and administration of medication, and importance of medication adherence. His caregiver would also require education on adherence to follow-up appointments, including those for well-checks and immunizations. Depending on the season (RSV season is typically from October/November through March/April, depending on geographic region), the patient may qualify for palivizumab for RSV

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prophylaxis, and thus his caregiver should be educated on the importance of follow-up for subsequent doses for the total course.

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CASE 5.1
Constipation | Level 1

Jeremy S. Stultz and Milap C. Nahata

1. What are the subjective and objective signs of constipation in this patient?

SUBJECTIVE FINDINGS: Difficulty forming stools for the past week, belly pain, fidgeting, and standing on his tiptoes

OBJECTIVE FINDINGS: Abdominal pain (6/10 on a visual analogue scale), mild abdominal distension, and tenderness with palpation

2. Classify this patient's constipation. What are the possible causes of constipation in this patient?

The constipation present in this child is likely functional (e.g., constipation with normal functioning bowels), but it may also have a minor organic (e.g., drug- or disease-related) component. This child is at an important and stressful developmental period in his life. Toilet training and starting pre-kindergarten is a big change, which can be difficult for some children. This situation can commonly lead to guarding and withholding bowl movements. Although this patient was toilet trained recently, it is still new to him. In addition, the patient had an "accident" recently at preschool. He may be unintentionally withholding stools due to some embarrassment from his previous "accident" and from the stress of toilet training and pre-kindergarten.

The diet reported from the mother also suggests that the patient may not be getting enough fiber in his diet (e.g., the patient does not like fruits, vegetables, or cereal). While this could implicate an organic etiology, it is less likely due to the presenting symptoms (i.e., tiptoeing and fidgeting) suggesting withholding.

3. Why is this patient a candidate for OTC treatment for his constipation?

The patient's constipation has not lasted more than a week and is likely functional in etiology. The mother has made verbal attempts to normalize the patient's bowel movements without success. Thus, a pharmacological intervention is warranted to maintain normal bowel habits. If a patient has constipation for >2 weeks, if there is noticeable fecal impaction, or if there are definitive organic causes (e.g., opioid use or structural abnormalities), then the patient should be referred to a physician for further evaluation.

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Without treatment, the constipation will likely continue and could lead to a chronic functional constipation where his normal reaction is to withhold stool when it is ready for defecation. Complications with chronic constipation include decreased appetite and consistent encopresis. Consistent withholding could cause frequent large, difficult to pass stools and even anal fissures. The patient should also receive some intervention to improve quality of life. The patient may be irritable, have difficulty sleeping, and not be able to attend pre-kindergarten. The mother may also have to take time out of her day (or job) to take care of the patient. The combination of pharmacologic and nonpharmacologic treatments is needed to resume normal bowel habits and ensure appropriate quality of life.

4. What nonpharmacologic treatment options are recommended for this patient?

The goal of therapy is to induce a bowel movement and then maintain bowel movements consistently. Nonpharmacologic options to induce a bowel movement include fluid consumption (water) and sorbitol-containing juices (e.g., 3 to 4 ounces/day of 100% prune juice, pear juice, and apple juice). This approach could be the first step to induce a bowel movement; however, there are limited data on their effectiveness. In this patient, these steps (e.g., daily apply juice intake) could be implemented after the first bowel movement to maintain consistent bowel movements.

In addition, the mother may also need to revisit toilet training in this patient with a focus on going to the bathroom when he needs to and not finishing until he has had a stool. She should also discuss the "accident" he had during school and reassure him that it is okay to ask to go to the bathroom in the toilet at school. This patient may experience pain with the first bowel movement as feces may have backed up during this period of constipation. A painful defecation could cause additional desire to withhold stools. The mother must reassure her child that it is not always painful and that right now he needs to "poop" even if it hurts a little because it will make his stomach feel better.

Other pertinent nonpharmacologic treatment options would be to increase fluid and fiber intake in the patient's diet. This can be done in many ways such as including cereals high in fiber (e.g., Raisin Bran®, Fiber One®, and All Bran® cereals), fruits, and vegetables in his diet on a daily basis. Fiber and appropriate fluid intake (~40 ounces of fluid per day) is important in maintaining stool consistency. Fiber supplements are available OTC, but use in children is not well studied.

5. What pharmacologic treatment options are recommended for this patient?

The pharmacologic agent of choice is dependent on the preferred route of administration and desired onset of action. Usually, the oral route is tried first, followed by rectal administration if bowel movements do not commence. Oral agents generally act slower than rectal agents but are better tolerated by the child.

A combination of docusate and sennoside treatment would be appropriate for this patient. Docusate sodium is a stool softener that would help loosen the stool in his colon, and sennoside is a stimulant laxative that will stimulate contraction of the gastrointestinal tract. Although docusate will likely help this patient, it takes longer to work (up to 72 hours for docusate versus within 24 hours for sennosides). Because he is in significant pain, a more effective treatment would be to combine docusate with a stimulant laxative. They come in various dosage forms. The combination product Senokot-S® (docusate sodium 50 mg and 8.6 mg sennosides per tablet, also usually available in generic forms) may be a reasonable choice for this patient if he can take a tablet. The appropriate dose would be ½ tablet once daily at night. The medication should be continued until the patient has a stool. After the patient has stooled, the sennoside can be discontinued, and docusate alone can be used until normal bowel habits have resumed (up to a maximum of 7 days). If a stool has not occurred within 3 days or normal bowel habits have not resumed within 7 days, the patient should be seen by a physician.

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Docusate sodium does come in a liquid dosage form in case the patient cannot take ½ of a tablet. Pedia-Lax® (docusate sodium 50 mg/15 mL) is available in most pharmacies. An appropriate dose in this patient would be 15 mL (50 mg) po daily. Some dosing recommendations suggest a dosing range of 20 to 60 mg/day divided into one to four doses. The directions on the OTC label suggest 1 to 3 tablespoons (50 to 150 mg) per day for patients 2 to 12 years of age. Although a sennoside syrup is commercially manufactured (8.8 mg/5 mL), it may not be available at all pharmacies and is not very palatable. An appropriate dose would be 2.5 mL (4.4 mg) at bedtime.

A pediatric glycerin suppository (glycerin 1.2 g suppository) is also an appropriate and effective treatment option (can work in 15 to 30 minutes). It should be given as one pediatric suppository rectally daily. This treatment can be offered to parents as an option, but it is usually not preferred due to the complicated rectal administration. It can instead be used if the oral medication(s) and nonpharmacologic options do not work after 3 days or the patient's pain continues to increase.

Alternative oral agents

- *Bisacodyl*: The OTC labeling for tablets does not provide recommendations for patients <6 years of age without recommending they see a physician, but the capsule formulation has dosing recommendations for patients >2 years of age. Use of bisacodyl capsules is an alternative stimulant laxative, but it is not generally preferred because it can cause significant abdominal cramping.
- *Polyethylene glycol:* Although this can be used in pediatrics and can be considered a first-line treatment, it is not suitable for OTC treatment in patients <16 years of age, and a physician consultation is warranted. The usual dosing is 0.5 g/kg/day and can be titrated to a maximum of 1.5 g/kg/day or 17 g/day. It is administered by adding the dose to 4 to 8 ounces of a beverage (e.g., water) before administration and ensuring the patient drinks the full amount of fluid.
- *Magnesium hydroxide and magnesium citrate:* These are alternative treatment options, but they are generally only used after other options have failed. Additionally, they should only be used under physician supervision. OTC labeling has recommendations only for patients 6 years and older.
- *Mineral oil:* This should not be used orally OTC in children under 5 years of age due to the risk of aspiration.

Alternative rectal agents

Rectal administration, enemas in particular, is unpleasant and difficult in children. Enemas can also lead to rectal bleeding if used improperly or excessively. Although these agents generally work faster (usually <30 minutes) and may be appropriate, they are usually not preferred first line. They can be recommended if patients are having severe pain or if they have not had a bowel movement despite 3 days of stool softener and stimulant laxative use. As described above, a glycerin suppository would be the first-line rectal treatment option for constipation. If a glycerin suppository is ineffective, the following are additional treatment options, although a physician consultation should be considered.

- *Sodium phosphate enema (also known as a "saline enema"):* OTC dosing is available for sodium phosphate enemas in patients ≥2 years. A 3-year-old would be given half a bottle of the pediatric enema. Only one dose should be given in a day; administering more than one dose will increase the risk of adverse reactions (e.g., electrolyte abnormalities).
- *Mineral oil enema:* OTC dosing is available for use of a mineral oil enema in patients ≥2 years. Similar to the sodium phosphate enema, a half bottle of a mineral oil enema can be used in this patient. This should also only be given as a single dose.
- *Bisacodyl enema and suppository:* This enema can be used in pediatric patients but only under the supervision of a physician. OTC recommendations are provided for the use of enemas only in patients 12 years and greater and suppositories in patients 6 years of age and greater; thus, this patient would need to see a physician before using this.

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6. Devise a pertinent patient education plan for the caregiver.

The mother should be educated on the effectiveness, safety, and appropriate use of the different OTC treatment options.

- *Effectiveness:* The mother should leave the pharmacy knowing that docusate and sennosides should be tried first. Sennosides should work in <24 hours, while the docusate may take up to 3 days. If the oral medications do not work after 1 to 2 days, then one glycerin suppository (1.2 g) can be tried. If there is still no relief, ½ of a pediatric sodium phosphate enema can be given, and a physician should be contacted. The suppositories and enemas should provide relief in <30 minutes.
- *Safety:* Oral docusate is usually well tolerated, but cramping and diarrhea can occur. Sennosides can also cause cramping, stomach pain, and diarrhea because it is a stimulant laxative that will stimulate the patient's gastrointestinal tract. It can cause more severe adverse reactions when it is used chronically (e.g., weeks to months), but chronic administration should not be recommended unless under the direction of a physician. Glycerin suppositories are generally well tolerated although uncomfortable for the patients. There are concerns of hyperphosphatemia and renal failure associated with phosphate enema use; however, these concerns are mainly due to the use of excessive enemas (e.g., more than one enema daily).
- *Appropriate use:* The mother should be instructed on how to split tablets if she chooses to administer tablets. If she prefers using a liquid preparation, she should be instructed on appropriate measurement of doses. Rectal administration techniques (of either a suppository or enema) should be described to her using the pictures on the back of the packages. If she desires to use the sodium phosphate enema or mineral oil enema, she should be instructed to take out half of the contents of the bottle before administering it. The nonpharmacologic treatments (e.g., fluids and fiber) should also be reinforced. Lastly, she should know that all of the pharmacologic treatments are for short-term relief of constipation. If the child is not able to have a bowel movement after 3 days of treatment or if bowel movements do not return to normal within a week after starting treatment (or 2 weeks of total symptoms), then she should take him to a physician for additional evaluation and treatment.

7. Identify any medication-related problems.

The use of NSAIDs should be discouraged during constipation, because it is not treating the constipation and may worsen his constipation. It should be verified if the patient has albuterol available at home, that it has not expired as it has not been refilled recently, and that the mother understands how to administer it. Additionally, the dose of IM epinephrine is too high for the patient's age and weight. The physician should be contacted, and it should be changed to the pediatric dose (0.15 mg).

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CASE 5.2
Crohn Disease | Level 2

Jeremy S. Stultz and Milap C. Nahata

1. What subjective and objective evidence suggests active CD in this patient?

SUBJECTIVE FINDINGS: Abdominal pain, fatigue, bloody diarrhea

OBJECTIVE FINDINGS: Abdominal tenderness and pain (8/10), underweight, appears fatigued, and appears mildly malnourished on PE; important laboratory tests: + guaiac stool test, leukocytosis, elevated ESR, elevated fecal calprotectin, anemia, hypocalcemia, hypoalbuminemia, and a Pediatric Crohn Disease Activity Index (PCDAI) of 42

The PCDAI score can be used to determine the severity of CD based on symptoms and laboratory data. The score uses a 0 to 100 scale, and a score of >30 indicates active moderate-to-severe disease. It is important to understand that this patient has chronic CD with an active flare-up.

2. What risk factors does this patient have for the development of active CD?

The potential risk factors for active CD in this patient are her age, sex, ibuprofen use, and race. Although age does not necessarily cause CD, the majority of CD diagnoses occur between 15 and 40 years of age. Females have a higher incidence of CD compared to males, although this is reversed in ulcerative colitis (UC). The connection between ibuprofen use and development of CD is controversial, but ibuprofen has been reported to acutely worsen CD disease and cause active disease. Her symptoms seem to have occurred before she started using OTC ibuprofen, but it may be worsening her active disease. Compared to those of African-American and Hispanic descent, Caucasians have a higher incidence of inflammatory bowel disease (IBD).

Psychosocial factors, mainly stress, have also been known to induce active disease in some patients. There is not enough information in this case to determine if stress is a contributing factor. Contraceptive use has been implicated to increase the risk of IBD, but reports are conflicting, and it appears to be higher when combined with smoking and when used for postmenopausal hormone replacement. Other accepted risk factors not present in this case include family history, level of physical activity (more physical activity decreases risk), and a history of smoking. Associations with CD have also been noted with various diets, nursing as an infant (breastfed infants may have less risk), antibiotic use, infections, obesity, and isotretinoin use for acne

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vulgaris, but these are not well-defined risk factors.

3. Assess the current pharmacologic management of this patient's CD.

The patient is currently exhibiting steroid dependent moderate-to-severe CD as indicated by the need for multiple steroid bursts this year and the resumption of symptoms while being tapered off the current steroid burst. Her disease has not been controlled with mesalamine. Various 5-ASA preparations have been used for treatment of mild-to-moderate CD. Although few studies have been completed in pediatrics, a meta-analysis of adult studies found that the role of 5-ASA remains unclear, and they are likely ineffective in severe disease.

Her steroid wean is not being tolerated, and she is having break-through symptoms. Budesonide is an alternative steroid with more localized action. Although fewer adverse reactions have been reported, studies have shown similar or less effectiveness compared to prednisone. Thus, switching to budesonide will likely not increase efficacy in this patient and is not a reasonable option. More aggressive CD treatment is warranted to induce and maintain remission. If an intervention is not made, multiple complications could occur. The patient may require long-term steroid treatment, which is not optimal treatment due to the numerous metabolic- and endocrine-related adverse reactions associated with long-term steroid use. Without adequate treatment, the extensive inflammatory disease could worsen and cause more severe complications such as fistulas and necrosis in her gastrointestinal tract. These complications may require extensive surgical management. She also will continue to have excessive bowel movements and will be malnourished, potentially requiring nutritional supplementation via parenteral nutrition.

The patient is *C. diff* toxin (-), and antibiotic therapy is not warranted for this patient's current presentation. Antibiotic therapy, usually ciprofloxacin or metronidazole, is often only used in patients with fistulas.

4. Develop a pharmacologic plan to induce remission for this patient's CD.

The goal of therapy in this patient is to induce a remission and then try to maintain it. Remission can be evidenced by a decreased number of bowel movements, absence of melena in her stools, decreased abdominal pain, decreased fatigue, improved oral intake, and appropriate weight gain. A decrease in the PCDAI score (overall disease severity) can also be used to determine a remission (scores of <10 have been used to define a remission). Initial treatment of CD is controversial and strategies have changed over the past decade. There has been a shift toward earlier immunomodulatory therapy after diagnosis. This patient may have been started on a 5-ASA agent at diagnosis because of a milder presentation. However, her current disease is more severe and thus requires more aggressive treatment to induce and maintain a remission.

An anti-tumor necrosis factor (anti-TNF) alpha agent would be an ideal treatment to induce remission in this patient. Recent studies (in both pediatric and adult populations) suggest earlier use of anti-TNF therapy after CD diagnosis leads to higher rates of remission, faster time to remission, and longer duration of remission compared to historical immunomodulatory treatments (e.g., methotrexate and azathioprine). Infliximab, although only FDA approved for induction and maintenance therapy of CD in children ≥6 years of age who failed conventional therapy, would be an appropriate treatment for this patient. An appropriate dose for this patient would be infliximab 200 mg (~5 mg/kg/dose) IV on weeks 0, 2, and 6 followed by 200 mg every 8 weeks. If treatment response is not achieved, the dose can be increased to 400 mg (10 mg/kg/dose) at the same frequency. Adalimumab has also been used to treat CD but is not approved by the FDA for use in pediatric patients, and data supporting its use in pediatrics are limited compared to infliximab. Although it may be beneficial in the ambulatory setting because it is administered subcutaneously and does not require hospital or infusion clinic admission for IV infusion, compliance can be a concern. This treatment should be reserved to failure with infliximab. Other biologics have

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minimal supporting evidence for use in pediatric patients.

The patient should receive the first dose of infliximab in the hospital. Once the patient is stable (no fever and improvement in bowel movements and abdominal pain allowing for adequate oral intake), she can be discharged with a follow-up when her next dose is due in 2 weeks. The patient was taking prednisone 10 mg po bid before admission. Due to the recurrence of symptoms, it may be beneficial to increase the dose of the prednisone while the infliximab is taking effect. Although the best dose is not well defined, a reasonable approach would be to reinitiate 20 mg po bid for 7 days, then decrease back to 10 mg bid for 7 days, and taper the dose as described in the previous prescription. This should allow the infliximab to start working while the steroids are weaned off. The titration can be increased or decreased based on symptom resolution. Mesalamine use should be reassessed once the disease undergoes remission. It can be used in combination with any of the above therapies, although efficacy is controversial.

Although not FDA approved for CD treatment, azathioprine and mercaptopurine have been used historically for the treatment of CD. Although effective at maintaining a remission, they exhibit a slow onset of action and are not optimal agents to induce a remission. Some clinicians may recommend one of these agents before infliximab based on experience with their use, but they are suboptimal choices for treatment in this patient as described previously. After this patient achieves remission, use of azathioprine or mercaptopurine may be considered for long-term maintenance treatment. Azathioprine dosing is usually 1 to 2 mg/kg/dose po once daily (IV available if needed), with doses as high as 4 mg/kg/day reported. If the patient is started on this treatment option, genetic testing should be performed to determine thiopurine methyltransferase activity. Slower metabolizers either require major dosage reductions or switching to an alternative therapy. The combination of a monoclonal antibody and azathioprine or mercaptopurine has been described and adult studies have shown possible enhanced efficacy. While promising, this treatment option can cause severe immunosuppression and is controversial. This treatment option should only be considered if monoclonal antibody treatment fails.

Methotrexate also has been used to maintain CD remission; however, it has a slower onset of action, similar to azathioprine, and significant toxicities. This should only be considered if the patient fails monoclonal antibody, azathioprine, and/or mercaptopurine treatment.

5. Offer potential interventions on medication-related problems for this patient.

Nonsteroidal anti-inflammatory drugs (NSAIDs) can be a gastrointestinal irritant through cyclooxygenase enzyme inhibition. Their use in IBD patients is controversial; some studies have suggested that they increase the risk of active disease, while other studies suggest they are well tolerated at lower doses for patients with musculoskeletal disease. In this patient's case there is not a clear benefit (e.g., treatment of uncontrolled musculoskeletal disease), which would outweigh the risk of adverse reactions. Ibuprofen should be discontinued, and she should be directed not to take any OTC NSAIDs. Acetaminophen can be used for mild pain at a dose of 500 mg po every 6 hours as needed.

Patients with CD are also at risk for nutritional deficiencies as well as vitamin and mineral deficiencies. If tolerated, protein-based nutritional supplements (e.g., Ensure®) could be considered to increase nutritional intake. Calcium supplementation should be started in this patient, because she has low serum calcium concentrations (8.2 after correcting for her albumin), and CD patients are at high risk for osteomalacia. A reasonable starting dose would be calcium carbonate 400 mg (elemental calcium) po 3 times daily. A vitamin D blood concentration (25-hydroxyvitamin D) should be obtained and vitamin D supplementation should be considered if blood concentrations are lower than normal. Vitamin D_3 (cholecalciferol) is generally preferred; the dosage regimen would depend on the serum concentration. The patient also currently has anemia. This is likely due to recent blood loss due to active disease. This

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should be followed and if still present when she is in remission, she should be worked up further for iron or vitamin B_{12} deficiency anemia (pernicious anemia), both of which can be seen in CD.

6. Include specific monitoring parameters for efficacy and safety of this patient's treatment.

Efficacy

The goal is to induce a remission with symptomatic and laboratory improvement. The PCDAI score can be calculated to determine if she is in remission, with <10 indicating disease remission. The patient should be instructed to keep a diary of her symptoms to accurately assess disease severity.

Repeat serum albumin at a follow-up visit in a month after discharge (track for improvement). Monitoring weight changes over time is also recommended.

Basic metabolic panel should be ordered at a follow-up visit to monitor for resolution of electrolyte abnormalities. Calcium, phosphorus, vitamin D concentration, magnesium, and iron concentrations should be checked periodically to ensure adequate nutrition.

Safety

This patient should be monitored closely while receiving her first dose of infliximab. Vital signs should be monitored during the treatments and nurses should be cognizant of any infusion-related reactions. Potential infusion-related reactions include chest tightness, shortness of breath, emesis, rash, fever, and blood pressure irregularities. Treatment of adverse reactions would be based on the symptoms (e.g., diphenhydramine for any rashes and acetaminophen for a fever). A tuberculin skin test should be performed to rule out latent or active tuberculosis before initiating treatment with infliximab because infliximab increases the risk of tuberculosis infections.

In general, all of the treatment options are immunosuppressive and potentially hepatotoxic. Vital signs and a CBC with a differential should be monitored at each infusion appointment. Specific toxicities with infliximab include anemia, neutropenia, and thrombocytopenia. Liver enzymes (e.g., AST and ALT) or liver function tests should be monitored to screen for potential hepatotoxicity. Concerns for malignancies have recently been reported with long-term use, and clinicians should be aware of this potential toxicity.

7. Suggest nonpharmacologic treatment options.

Diet can play a role in the management of CD. Many different foods and drinks have been implicated as causing disease flares. Some common ones are alcohol, spicy foods, coffee, tea, chocolate, foods high in fiber, some raw fruits and vegetables, and red meat or pork. Specific dietary triggers may be different for each patient. It should be recommended that the patient maintain a diary of the different foods and drinks she consumes to determine what causes acute worsening of her disease. These foods should be avoided in the future.

The patient also has been recently diagnosed with depression. Dealing with a chronic disease like CD can be very difficult for adolescents emotionally and is likely a major component of her depression. The patient should be given information regarding any local support groups for patients with CD.

8. Recommend appropriate immunizations for this patient.

This patient should receive an annual influenza vaccine. This patient should only receive the inactivated influenza vaccine; the intranasal live vaccine is contraindicated in this patient as she is on immunosuppressive therapy. She is also a candidate for the pneumococcal vaccine. This patient has not received any pneumococcal vaccinations before (the 7- and 13-valent vaccines were not yet available). Based on the 2014 CDC immunization recommendations, she should receive a single dose of the 13-valent vaccine followed by the 23-valent pneumococcal vaccine, with 8 weeks in between doses to ensure adequate immunization.

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CASE 6.1
Nephrotic Syndrome | Level 2

Sandra Benavides

1. What are the subjective and objective signs and symptoms of nephrotic syndrome in the patient?

SUBJECTIVE FINDINGS: The patient presented to the nephrology clinic with subjective signs of nephrotic syndrome including appearing swollen with mild abdominal discomfort and leg edema.

OBJECTIVE FINDINGS: The patient has an extensive 10-year medical history of nephrotic syndrome with multiple relapses. His blood pressure is elevated at 138/94. On physical exam, he has 2+ pitting edema on both legs and ascites. His laboratory analyses indicate a low serum albumin, an elevated urine protein, and microalbumin. He also has a low vitamin D serum concentration. The patient has elevated protein in his urine; however, his urine protein/creatinine ratio is 0.14 indicating he is in remission at this time. Lastly, the kidney biopsy is positive for focal segmental glomerulosclerosis.

The patient has been diagnosed with frequently relapsing, steroid-dependent nephrotic syndrome of which he is in remission based on his urine protein/creatinine ratio of less than 0.2. He is considered to be frequently relapsing because he has had more than four relapses in a year's time. He has also had a history of multiple relapses within 14 days of discontinuation of corticosteroids, indicating he has steroid-dependent nephrotic syndrome. His biopsy confirms a diagnosis of focal segmental glomerulosclerosis.

2. Describe the treatment goals and outcomes in the patient.

Treatment of nephrotic syndrome is directed at reducing symptoms and preventing complications, including end-stage renal disease (ESRD). During a relapse of nephrotic syndrome, patients may experience edema secondary to low albumin and decreased oncotic pressure in the intravascular space. Electrolyte abnormalities, including hyponatremia, may also be present. Dyslipidemia, manifested by increased total cholesterol and triglyceride concentrations, is common during active nephrotic syndrome disease. In a relapse of nephrotic syndrome, restoration of the intravascular volume and normalization of total cholesterol and triglycerides are the primary goals.

Long-term complications associated with nephrotic syndrome include ESRD, recurrent viral infections, and thromboembolic events. These are directly related to nephrotic syndrome whereas other complications are related to the treatment of the disease, such as cardiovascular

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disease, opportunistic infections, osteopenia/osteoporosis, and hypothyroidism. Prevention of a relapse and complications of nephrotic syndrome is the long-term treatment goal. Additionally, appropriate diet to achieve normal growth and development is important.

3. Evaluate the appropriateness of each treatment strategy for nephrotic syndrome in the patient and recommend any necessary changes to his regimen.

Treatment options for frequent relapsing or steroid-dependent nephrotic syndrome include oral cyclophosphamide (2 mg/kg per day for 12 weeks, maximum cumulative dose of 168 mg/kg), mycophenolate mofetil (24 to 36 mg/kg/day, maximum 2 g per day), or cyclosporine A (3 to 5 mg/kg/day divided twice daily). The patient has failed therapy with cyclosporine because he had an episode of relapse while on the medication. He is currently on mycophenolate 500 mg po bid (approximately 13 mg/kg/day). His dose can be increased to 1,000 mg po bid (approximately 26 mg/kg). However, his current white blood cell (WBC) count is low and should be monitored. If the WBC count recovers, he should continue on the mycophenolate for a year's time if he remains in remission for 6 months. If he is not in remission at 6 months or if he is unable to tolerate mycophenolate, cyclophosphamide at 150 mg daily for 12 weeks would be the next medication to administer. However, adherence to medications must be assessed to determine whether treatment failure was a result of an ineffective drug or nonadherence.

The corticosteroid should be continued long term due to relapses; however, the dose should be tapered down to the lowest dose to control symptoms and minimize adverse reactions. Because the patient is currently in remission, the current prednisone dose of 60 mg (2 mg/kg, maximum 60 mg) can be decreased to 40 mg (1.5 mg/kg, maximum 40 mg) every other day for 4 weeks. If tolerated, the dose can be further decreased to 40 mg every other day for 2 weeks, 30 mg every other day for 2 weeks, 20 mg every other day for 2 weeks, to a goal of 10 mg every other day. If at any time during the taper symptoms reappear, the dose may have to be increased to the previously effective dose.

An angiotensin-converting enzyme inhibitor or an angiotensin receptor blocker is the first-line therapy in patients with hypertension and has been shown to prevent future kidney disease in patients with proteinuria. The patient is on lisinopril 10 mg daily. The patient is not at goal blood pressure, which should be less than the 90th percentile for age, gender, and height percentile (i.e., less than 138/83). Before increasing the dose of the lisinopril, it is important to assess adherence. If the patient is taking the medication as prescribed, the dose can be increased to 20 mg today, then by 10 mg at a 1- to 2-week interval, to a maximum dose of 40 mg/day. If the patient begins to develop a cough associated with the lisinopril, an angiotensin receptor blocker is an alternative choice with renal protective benefits. Efficacy is monitored by measuring the patient's microalbuminuria. Values less than 300 mcg/mL are considered to be controlled in patients with nephrotic syndrome.

Dyslipidemias are common in uncontrolled nephrotic syndrome often requiring the use of an HMG-CoA reductase inhibitor. When nephrotic syndrome is in remission, lipoproteins tend to normalize. Treatment guidelines for dyslipidemias recommend pharmacotherapy when LDL is greater than 160 mg/dL in patients with nephrotic syndrome and an additional moderate or high-level risk factor (such as hypertension requiring pharmacotherapy, obesity, family history, or smoking). Because the patient's fasting lipid panel was in normal range, the simvastatin can be discontinued. Another fasting lipid panel should be obtained in 6 months or if the patient undergoes a relapse. It is important to educate the patient on lifestyle modifications and the importance of a low-fat, low-cholesterol diet.

The patient is experiencing edema and requires 20 mg furosemide daily. Because he is still experiencing signs of edema (as evidenced by the 2+ pitting edema to knees and ascites), he may benefit from an increase in the dose to 40 mg once daily (to a maximum of 80 mg if needed). Because of the nonadherence to medications, the dose should be given as one

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daily dose rather than 20 mg twice daily in an effort to improve adherence.

4. Identify and resolve the medication-related problem(s).

The patient may be experiencing leukopenia secondary to mycophenolate. The WBC count should be monitored closely by obtaining a CBC weekly. If the WBC count falls with an absolute neutrophil count (ANC) less than 1,500, the medication should be held until the restoration of the WBC count and ANC. Another agent (cyclophosphamide) may be considered as an alternative treatment.

Patients with nephrotic syndrome should avoid nonsteroidal anti-inflammatory drugs (NSAIDs) if possible. NSAID use in the face of nephrotic syndrome can precipitate acute kidney injury secondary to the prostaglandin synthesis inhibition via the cyclooxygenase II enzyme inhibition. In patients with nephrotic syndrome, prostaglandin synthesis may be elevated secondary to renal vasoconstriction. The elevated prostaglandins serve to maintain the glomerular filtration rate. The patient should be counseled to discontinue the use of the ibuprofen (and avoid any other NSAIDs) and use acetaminophen for any pain or fever.

5. Describe short-term and long-term adverse reactions to corticosteroids and management of adverse reactions.

Initial adverse reactions to glucocorticoid therapy include increased appetite, weight gain, gastrointestinal disturbances, mood abnormalities, increased blood pressure, leukocytosis, and hyperglycemia. Patient counseling should include instruction about taking medication with food and the potential for anxiety or insomnia related to steroid therapy. During a hospitalization, daily monitoring of blood pressure, blood glucose, and weight is important in this patient.

There are a number of possible long-term adverse reactions to corticosteroids. Once the patient requires the use of systemic corticosteroids for more than 2 weeks, monitoring and identification of toxicities is necessary. These include hypothalamic-pituitary-adrenal (HPA) axis suppression, growth suppression, immunodeficiency, and decreased bone mineralization resulting in osteopenia or osteoporosis, cataract formation, and glaucoma.

HPA axis suppression can occur as a result of long-term exogenous administration of corticosteroids due to the negative feedback to the hypothalamus subsequently stopping the release of cortisol from the adrenal glands. If corticosteroids are abruptly discontinued after long-term use, the patient may develop adrenal crisis or insufficiency if he encounters a stressful situation (e.g., surgery, trauma, illness). Both adrenal crisis and insufficiency can be life threatening if not recognized or treated immediately. The patient must be reminded to not stop the medication abruptly. Because he has been on long-term corticosteroids, he may benefit from a medical alert bracelet in the event of an emergency.

Growth suppression occurs in children who require long-term systemic or high-dose inhaled corticosteroids. Because the patient is on long-term systemic corticosteroids, his height and weight should be monitored at least twice a year. In this patient, it may be helpful to obtain a bone age of the hands and wrists to determine if he has reached his full adult height potential. To minimize the effects, the dose of corticosteroids should be started at the lowest dose possible to control the disease and administered once a day or once every other day if possible. The patient is between the 50th and 75th percentile for height, which is above average for his age and gender.

The immunosuppressive effects of corticosteroids increase the risks of gram-negative, fungal, and herpes virus infections as well as reactivation of tuberculosis infections. Any signs of infection warrant an immediate evaluation and management.

Osteopenia and osteoporosis leading to fractures is a severe adverse reaction of long-term corticosteroid use. The risk is highest within the first 6 to 12 months of use. The patient should obtain a bone mineral density examination (if he does not have a baseline one) and repeat the exam annually. Also, he should receive vitamin D and calcium supplementa-

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tion. The vitamin D serum concentration is below normal limits and therefore requires a higher dose of 1,000 international units daily (as opposed to the recommended dose of 400 international units for supplementation in those taking corticosteroids). Calcium should also be supplemented. He should receive 1,500 mg of calcium daily. Weight-bearing exercise would also assist in improving and maintaining normal bone mass.

Eye complications due to corticosteroid include cataracts and glaucoma. The patient should obtain an annual eye exam while on corticosteroids. Lastly, blood pressure and a basic metabolic panel should be monitored at least every 6 months while on corticosteroid therapy.

6. List other patient counseling points important to communicate with the caregiver and patient.

a. Adherence to treatment regimen should be emphasized to the patient and caregiver at each visit to avoid relapse of nephrotic syndrome.

b. The patient should be on a low-fat, low-sodium diet. Because the patient is at increased risk for dyslipidemia when the nephrotic syndrome is not controlled, the patient may be at high risk for cardiovascular disease. The diet should contain no more than 30% of fat, less than 10% of saturated fat, and less than 300 mg/day of cholesterol. His sodium should be restricted to 1,500 to 2,000 mg daily to minimize edema.

c. Patients with nephrotic syndrome are at high risk for infections. Patients should be immunized with the pneumococcal polysaccharide vaccine (PPSV23). If the patient had never received the pneumococcal conjugate vaccine (PCV7 or PCV13), he would require administration at this time. However, the patient's immunization status is currently up to date, which implies he has received the vaccine. If the patient has received the PPSV23, he will require one booster at least 5 years later for a maximum of two in his lifetime. The patient and all household contacts should receive the annual inactivated influenza vaccine. The patient should not get any live vaccines until 1 month after the discontinuation of immunosuppressant agents (mycophenolate).

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CASE 6.2
Acute Kidney Injury | Level 3

Jeremy S. Stultz and Milap C. Nahata

1. Identify any potential causes of AKI in this patient.

There are numerous risk factors for AKI in this patient, and the cause is likely multifactorial. Shock and hemodynamic compromise have likely caused initial hypovolemia (elevated BUN/SCr ratio) and prerenal injury. Despite the administration of fluid, septic shock has progressed with impaired circulation and decreased renal perfusion (evidenced by elevated lactate concentration), likely leading to ischemic acute tubular necrosis. Norepinephrine, while the best treatment option for patients with warm septic shock, can also decrease perfusion to the kidneys due to vasoconstriction. This likely enhanced the acute tubular necrosis that was occurring.

Gentamicin is also commonly associated with acute tubular necrosis (similar to the other aminoglycosides). It displays saturable binding to the brush-border membrane of the proximal tubular cells, and accumulation can cause direct proximal tubule damage. This is a potential cause; however, there have been no serum concentrations measured so far and renal toxicity occurs more frequently with elevated concentrations and longer treatment durations. The role of vancomycin in renal failure is widely debated; however, it is commonly associated with nephrotoxicity when combined with other risk factors for nephrotoxicity (e.g., shock and concomitant nephrotoxic agents). As with gentamicin, serum concentrations have not been assessed at this point. Although there were signs of dehydration on admission and there was likely some renal compromise, her SCr was only mildly elevated (although delays in SCr increases are common in AKI), and thus the initial dosing was appropriate for the given information.

The combination of these issues puts this patient at high risk for AKI.

2. Identify the subjective and objective evidence suggesting AKI in this patient.

SUBJECTIVE FINDINGS: None pertinent to AKI in this patient

OBJECTIVE FINDINGS:

- Elevated BUN and serum creatinine (ratio 55:1 on admission, 27:1 now)—This illustrates hypovolemia on presentation with some improvement after volume resuscitation. The ratio is still elevated above 20, suggesting some prerenal toxicity may still be occurring.
- UOP <0.5 units/kg/hr over the past 12 hours (constant decrease)

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- Fluid accumulation evidenced by weight gain (positive 1.6 kg)
- High anion gap metabolic acidosis (decrease CO_2, low pH, and high anion gap)
- Mild hyperkalemia (suggests some inability to appropriately remove potassium via the kidneys)
- Urinalysis with WBC count, RBC count, protein, casts, and a fractional excretion of sodium of 2.1% ([Urine Na × SCr]/[Urine Cr × serum Na] × 100)—All indicate ATN is present.

3. Assess AKI based on creatinine clearance, the pRIFLE criteria, and UOP.

The patient has a baseline SCr of 0.42 mg/dL. Using the bedside Schwartz equation (the appropriate equation when using the isotope dilution mass spectrometry [IDMS] traceable SCr assay), her CrCl can be estimated at 123 mL/min/1.73 m^2. On admission, the SCr was increased to 0.51 mg/dL, an estimated CrCl of 101 mL/min/1.73 m^2. This clearance estimate was only 17.8% decreased from her baseline and would not be considered AKI based on the pediatric risk, injury, failure, loss, and end-stage renal disease (pRIFLE) critieria.

After 36 hours, the SCr increased to 1.2 mg/dL. The approximate CrCl would be 43 mL/min/1.73 m^2 (65% decrease from baseline). The patient would currently be in the "I" (injury) category of the pRIFLE criteria. However, SCr is a delayed marker of renal function and, as such, this concentration will likely continue to rise over the next few days. The patient would go into the "F" (failure) category of pRIFLE if the CrCl goes below 35 mL/min/1.73 m^2 (SCr >1.4, a decline of greater than 75% from baseline).

UOP is an important marker for kidney function, especially in pediatric patients. This patient's UOP was 0.54 mL/kg/hr over the past 24 hours and 0.35 mL/kg/hr over the last 12 hours. This again places the patient in the "I" (injury) category of pRIFLE. If the UOP continues to decrease (<0.3 mL/kg/hr over 24 hours), she would be classified as "F" or renal failure. When categorizing a patient based on UOP and SCr changes, the worse marker between UOP and SCr would determine the category in which the patient would be placed. Although the use of pRIFLE and other AKI definitions, like the Kidney Disease Improving Global Outcome AKI categories, can provide a means of consistency for both clinical practice and research, not all patients need to be categorized by these definitions. They are generally used as a reference and the combination of UOP, SCr, fluid balance, and electrolyte balance trends over time are commonly used to estimate renal function.

4. Identify the management goals for this patient's AKI.

The main goals are to prevent further renal damage, prevent complications from renal insufficiency, and avoid dialysis. Common complications of AKI include fluid overload, electrolyte abnormalities, and toxicity due to excessive drug exposure if dosage is not adjusted. Pharmacists can play an important role by performing risk versus benefit assessments of nephrotoxic agents, adjusting medication dosing for medications excreted via the kidneys, and selecting appropriate dosage forms to prevent excessive fluid accumulation. Each medication (including IV fluids) should be checked to determine potential ways to prevent or manage AKI-related complications.

5. Create a pharmacotherapeutic plan for managing this patient while she has AKI. Offer potential interventions on any medication-related problems to ensure safe and effective treatment of this patient while she has AKI.

It is important to note that there is usually not one specific treatment for AKI; instead multiple interventions are made to manage patients during their AKI and prevent progression and complications. Renal replacement therapy is not required at this point because there is currently only renal injury, mild metabolic and electrolyte abnormalities (without symptoms), and the patient continues to have some UOP. Close monitoring of these parameters should be used, and a nephrology consult should be placed in case dialysis is eventually needed. Perti-

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nent pharmacotherapeutic plans are discussed below.

MINIMIZING NEPHROTOXIC MEDICATIONS ON EXPOSURE

a. Although norepinephrine may be decreasing renal perfusion, it is the vasopressor of choice for warm septic shock, which occurs in ~20% of pediatric septic shock patients ($ScvO_2$ has been >70% during shock state). The current hemodynamic parameters suggest her blood pressure has stabilized even after weaning the norepinephrine to 0.1 mcg/kg/min, and her shock is slowly resolving. This wean should be continued (assessments every 30 minutes to 1 hour) with a goal of discontinuing the norepinephrine as soon as possible.

Dopamine or epinephrine are alternative agents and are better for patients in cold shock, but they do not have the same vasopressive effects needed for a patient in warm shock and provide excessive inotropic and chronotropic activity on the heart that is not needed in warm shock. Dopamine, starting at 5 mcg/kg/min, could be used to facilitate the norepinephrine wean if the patient is unable to titrate off norepinephrine. However, doses >15 mcg/kg/min will also have strong vasopressive effects and will decrease kidney perfusion the same as norepinephrine. Dobutamine is not generally recommended in septic shock, especially warm shock, because it has no effect on vascular resistance.

b. The blood cultures are currently growing gram-positive cocci in clusters; however, the results are preliminary. The patient failed third-generation cephalosporin treatment (although orally administered), suggesting it may be methicillin-resistant *Staphylococcus aureus* (MRSA) rather than methicillin susceptible. Vancomycin should cover most gram-positive cocci organisms and is still considered a first-line treatment option. However, it has been associated with nephrotoxicity and should only be continued with close serum concentration monitoring. The next dose of vancomycin should be held and a serum trough concentration measured from a sample (drawn via peripheral venipuncture) 5.5 to 6 hours after the last dose. The trough is likely elevated and the frequency of vancomycin dosing will need to be adjusted based on the serum concentration (e.g., every 6 hours to every 12 hours or every 24 hours). If the blood/sputum cultures come back as a gram (+) organism species susceptible to alternative treatments, the regimen should be tailored based on those culture results.

Alternative options to vancomycin with adequate gram (+) coverage (e.g., MRSA coverage) are not very robust for this patient case. The patient could be switched to linezolid 250 mg (10 mg/kg/dose) IV every 8 hours to avoid any potential renal toxicity from vancomycin. Linezolid has been well established in the treatment of pneumonia. However, linezolid is reported to be more bacteriostatic than bacteriocidal against MRSA (although controversial). Bacteriocidal agents are preferred for treatment of septic shock, but this patient's septic shock is starting to resolve. If the renal function continues to decline and her septic shock continues to resolve (i.e., she is able to come off vasopressor support), linezolid should be initiated. Total treatment should be 10 to 14 days after the first negative blood culture (or more based on clinical response). Linezolid does not require dosage adjustment for renal insufficiency, but use of this agent long term (>28 days) can have significant toxicity (e.g., lactic acidosis, peripheral neuropathy, thrombocytopenia, and irreversible ocular toxicity). Clindamycin can be added to vancomycin to prevent potential community-associated MRSA (CA-MRSA) toxin production at a dose of 250 mg (10 mg/kg/dose) IV every 8 hours, but it should not be used as a single treatment in this patient because it is a bacteriostatic agent and MRSA susceptibility is variable. This intervention is mainly based on in vitro data, and its impact on clinical outcomes has not been well described.

Daptomycin is not recommended in this case because the patient has pneumonia and daptomycin is inactivated by lung

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surfactant. Tigecycline and quinupristin/dalfopristin have been used to treat gram (+) infections after failure with vancomycin in adults without reports of causing renal insufficiency or requiring renal dosage adjustments. Data regarding use of these agents in pediatrics are limited and should only be considered if other treatment options have failed. Ceftaroline does not have data yet available for use in pediatric patients and cannot be recommended at this time. Other anti-MRSA agents (e.g., doxycycline, sulfamethoxazole–trimethoprim) have variable susceptibility patterns and are not optimal agents.

c. A gentamicin serum concentration (drawn via a peripheral venipuncture), 7.5 to 8 hours after the last dose, should be obtained. Continuation of gentamicin with dosage adjustments based on serum concentrations is controversial as it could worsen renal insufficiency. If the culture results are finalized as MRSA, some experts will recommend continuing low dose gentamicin (e.g., 1 mg/kg/dose) for synergy against MRSA. This could be done with close monitoring of serum concentrations to avoid any drug accumulation but is not a standard recommendation.

Alternatively, to ensure adequate gram (–) coverage (e.g., for *Pseudomonas aeruginosa*) until culture results are final and to prevent renal toxicity, gentamicin should be discontinued and switched to a fluoroquinolone. Reasonable regimens would be ciprofloxacin 375 mg IV (15 mg/kg/dose) every 12 hours or levofloxacin 250 mg (10 mg/kg/dose) IV every 24 hours. If kidney function worsens to <30 mL/min/1.73 m^2, the dosages should be decreased. There are FDA warnings about adverse reactions related to joints and surrounding tissue development with the use of fluoroquinolones in pediatric patients, most of which comes from cartilage erosion noticed in immature animals. The potential benefits of fluoroquinolone use in this case far outweigh the risk because sepsis is a life-threatening disease.

Meropenem is appropriate in this case to provide double coverage for resistant gram (–) organisms (e.g., *Pseudomonas aeruginosa*) because these organisms are not 100% susceptible to aminoglycosides and fluoroquinolones and can be difficult to treat with monotherapy. The routine practice of double coverage is controversial and should be based on local susceptibility patterns. The use of meropenem in this patient is also appropriate due to the severity of illness and the treatment failure with a third-generation cephalosporin. Other options are also reasonable (e.g., ceftazidime, cefepime), but it is important to remember double coverage should not include two drugs with similar mechanisms of actions. For example, cefepime and meropenem should not be used together, as they are both beta-lactam antibiotics. Because the patient has renal insufficiency, the dosage should be decreased to 1,000 mg IV every 12 hours. If renal function worsens, the frequency should be reduced further. If the cultures are finalized as a gram (+) organism, broad-spectrum gram (–) coverage can be discontinued.

FLUID AND ELECTROLYTES

a. The patient requires pharmacologic diuresis due to current fluid overload of approximately 10%, which can be determined with the following equation:

[fluid in (L) − fluid out (L)]/admit weight (kg) × 100

A loop diuretic is a good choice to remove excess fluid and may help correct the mild hyperkalemia. Furosemide can be given at 2 mg/kg (50 mg) once and then if a diuretic response is noted, continued either every 6 hours or via continuous infusion starting at 0.05 mg/kg/hr. The use of loop diuretics has not been shown to alter the course of renal injury, but it has been suggested that the percent of fluid overload before renal replacement therapy may be associated with mortality in severely ill children. Fenoldopam and low dose dopamine (<3 mcg/kg/min) are other agents commonly used to promote diuresis in critically ill pediatric patients, but again they have not been shown to alter the course of renal

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injury. There are some studies suggesting decreased immune system and pituitary function with the use of dopamine in septic shock, and thus some clinicians will recommend fenoldopam over dopamine. However, significant cost differences make it difficult to recommend fenoldopam over dopamine without definitive clinical benefit.

b. The patient's glucose is highly elevated and above the renal threshold of ~180 mg/dL, resulting in glycosuria. An insulin drip should be initiated to both help with carbohydrate utilization and to prevent further spilling of glucose in the urine. An appropriate starting dose would be insulin regular (1 unit/mL in NS) IV at 2.5 mL/hr = 0.1 units/kg/hr. This can be titrated every hour based on glucose concentrations with a goal serum glucose concentration of 60 to 180 mg/dL. The kidneys eliminate insulin, thus it should be titrated cautiously to prevent overcorrection. Dextrose-containing fluids should always be given while insulin is being administered continuously in case hypoglycemia occurs.

c. To avoid intermittent glucose boluses, meropenem and vancomycin could be made in NS instead of D5%W. The norepinephrine diluent cannot be changed to NS as D5%W is needed to protect against oxidation.

d. As this patient is currently fluid overloaded, the maintenance fluid rate should be decreased to at least 75% of maintenance (about 50 mL/hr) and adjusted as needed. Additionally, continuous and intermittent IV medication concentrations should be maximized. Fentanyl can be given undiluted (50 mcg/mL), meropenem can be reconstituted as 50 mg/mL, and norepinephrine can be made as a 64 mcg/mL solution. This will significantly decrease the amount of fluid from norepinephrine in this patient. The highest concentration of norepinephrine has not been well studied, and some institutions use more concentrated infusions. Caution should be used because it is a vesicant and could cause significant tissue damage when administered at higher concentrations (it should not be given peripherally). Stability data are also not readily available for use of higher concentrations for longer periods of time.

e. Potassium should be removed from the maintenance IV fluids due to the increasing serum potassium.

f. Based on serum CO_2 measurements, prn orders for sodium bicarbonate could be used to treat metabolic acidosis. Alternatively, sodium acetate or bicarbonate could be added to the IV fluids to treat metabolic acidosis.

g. If the current trophic feeds (i.e., Pedia-Sure® at 10 mL/hr) are not well tolerated (e.g., abdominal distension) with plans to increase enteral feedings, a total parenteral nutrition consultation should be considered.

6. Devise appropriate monitoring parameters for management of this patient.

a. Vital signs should be checked every 15 to 30 minutes. Goal BP would be above the bottom 5th percentile of BP for her age (~95/57 mm Hg). Norepinephrine should be weaned as long as the BP remains above that range. Her HR has returned to normal and should still be monitored. Temperature should be monitored at least 4 times daily to evaluate for infection resolution. Analgesia and sedation scores should be monitored hourly with adjustments to fentanyl and midazolam as needed.

b. Weight should be recorded daily, but the admission weight should be used as the dosing weight as she is currently fluid overloaded. I/O should be recorded by the nursing staff hourly and reviewed frequently each day. Goal UOP should be >1.0 mL/kg/hr. The use of diuretic therapy could cause additional hypotension, especially at higher doses, so BP should also be monitored.

c. A complete metabolic panel should be ordered at least daily with arterial blood gases scheduled at least every 4 hours (ionized calcium, potassium, glucose, and lactate can be added to these labs) as needed to make ventilator adjustments and to determine treatments for metabolic acidosis, hyperglycemia, and hyperkalemia.

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i. Specific attention should be paid to electrolytes and minerals that are eliminated via the kidneys, such as potassium, magnesium, calcium, and phosphorus. Fluids and prn orders should be adjusted as needed.

d. A CBC should be obtained at least every other day to monitor for resolution of infection (decreases in WBC count) and potential drug toxicities (e.g., platelets if linezolid is initiated).

e. Peripheral and central-line cultures should be repeated daily until the cultures do not grow any organisms. The first day of negative cultures should count as the first full day of antibiotic treatment.

f. Cardiac telemetry should be monitored as this patient has mild hyperkalemia.

g. If vancomycin or gentamicin treatment is continued, dosages should be adjusted based on serum concentrations to ensure adequate clearance. Goal concentrations are vancomycin, trough, 15 to 20 mcg/mL; gentamicin, peak 8 to 10 mcg/mL, trough <2 mcg/mL; gentamicin synergy, peak 3 to 5 mcg/mL, trough <0.5 mcg/mL.

h. If the patient becomes anuric despite furosemide treatment, exhibits hyperkalemia despite furosemide or other diuretic treatment, or has worsening metabolic acidosis, renal replacement therapies (e.g., hemodialysis) should be considered.

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CASE 7.1
Type 2 Diabetes Mellitus | Level 1

Sandra Benavides

1. What is the subjective and objective evidence for the diagnosis of T2DM?

SUBJECTIVE FINDINGS: The patient did not report any subjective findings of T2DM as he denied polydipsia, polyuria, and fatigue. He may be experiencing polyphagia as he tends to overeat and has large portion sizes, but this appears to be an ongoing issue from before the onset of diabetes.

OBJECTIVE FINDINGS: The objective evidence of T2DM includes fasting plasma glucose of 185 mg/dL and HbA_{1c} of 8.2%, which satisfy diagnostic criteria for diabetes. Although islet cell antibodies were not obtained in this patient, there are indications that he has T2DM such as elevated fasting insulin and C-peptide, acanthosis nigricans (a symptom of insulin resistance), morbid obesity, strong family history, and he belongs to an ethnic group that has a high risk for T2DM.

2. Assess the need for therapy for dyslipidemia and prehypertension for this patient.

The complications of T2DM include microvascular and macrovascular disease. Microvascular complications include retinopathy, nephropathy, and neuropathy, whereas macrovascular complications include peripheral, cardiovascular, and cerebrovascular diseases. Other complications can include recurrent infections. Because the onset of T2DM in children is a relatively new phenomenon, little data exist on the natural history of the disease. However, several studies have reported an earlier onset of microalbuminuria and higher risk of kidney disease compared to children with type 1 diabetes. One study predicts that children diagnosed with T2DM early in life can expect a 15-year reduction of life and advanced complications by the age of 40. The patient should be treated to prevent complications of diabetes and early mortality.

Because diabetes is a risk factor for cardiovascular and cerebrovascular disease, the management of dyslipidemia or hypertension is important. The patient should have two fasting lipid profiles separated by at least 2 weeks but no more than 3 months because he is considered high risk for cardiovascular disease due to the diagnosis of diabetes and obesity. He may also be at risk secondary to a strong family history, but further information must be gathered including if any family member has had a history of premature vascular disease (e.g., stroke, myocardial infarction). Nonpharmacologic treatment

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strategies should be employed to improve the lipid profile as treatment with pharmacologic therapy is not indicated until the LDL concentrations are greater than 130 mg/dL.

The patient is currently classified as having prehypertension as his current blood pressure is between the 90th and 95th percentile for his height, age, and gender. The diagnosis of hypertension requires repeated accurate measurements of blood pressure (with an appropriately sized cuff) or with ambulatory blood pressure monitoring. Pharmacologic therapy would be indicated if he is prehypertensive because of his diagnosis of T2DM. In addition, an echocardiogram should be considered to determine if left ventricular hypertrophy (LVH) (evidence of target organ damage) is present.

3. Develop a pharmacologic regimen for the treatment of T2DM.

Food and Drug Administration (FDA) approved treatment options for T2DM in children and adolescents are limited to insulin and metformin. In this patient, the drug of choice is metformin because his blood glucose is less than 250 mg/dL and his HbA_{1c} is less than 9%. In patients that exceed the above values, insulin is preferred in order to gain glucose control more rapidly. Benefits to using insulin initially may allow the pancreas to rest from the overproduction of insulin and may lead to improved compliance in the child or adolescent long term because of the perceived gravity of a disease that requires daily injections.

Although lifestyle modifications will have to be addressed, it is recommended to initiate therapy with metformin at diagnosis due to the challenges related to diet and exercise in the adolescent population. The dose should be initiated at 500 mg po daily, increasing by 500 mg on a weekly basis (if not tolerated, every 2 weeks) up to a maximum dose of 1,000 mg twice daily. Little efficacy has been noted in doses greater than 2 g per day. The benefits of metformin include proven efficacy of decreasing the HbA_{1c} by 1% to 2%, potential weight loss and improvement of lipids, and safety profile. The goal for the patient would be an HbA_{1c} less than 7%. His fasting serum glucose concentrations should be between 70 and 130 mg/dL.

The patient should have an HbA_{1c} monitored every 3 months. He should also be instructed to do self-monitoring of blood glucose (SMBG) before meals (including a fasting AM) and before bedtime to determine efficacy of the metformin and lifestyle modifications. Once the patient achieves the therapeutic goals, the frequency of testing can be decreased as metformin monotherapy does not cause hypoglycemia. Prior to initiation of therapy and then yearly, a serum creatinine, hepatic function tests, and a CBC should be obtained. Adverse reactions related to metformin include diarrhea, which is minimized with a slow titration of the agent and decreases with time.

If the patient is unable to achieve control with metformin monotherapy, the addition of long-acting insulin (e.g., glargine) nightly may be necessary.

4. Determine an appropriate treatment strategy for morbid obesity in this patient.

The patient must be counseled on diet and exercise for the treatment of obesity, diabetes, and improvement of his LDL and HDL cholesterol. He is considered to be morbidly obese as his BMI falls above the 99th percentile for his sex and age. Because he also has comorbid conditions (diabetes), a weight-loss treatment plan is indicated. He would benefit from a structured weight management program, comprehensive multidisciplinary intervention, or tertiary center intervention based on program availability. The patient and family should be referred to a nutritionist to discuss healthy food options, if available.

Ideally, the patient should restrict his caloric intake to 1,200 calories per day with less than 30% of calories from fat. However, this may be a challenging goal. Some strategies to decrease caloric intake is to eat regularly scheduled meals and snacks, limit portion sizes, eliminate soda from diet, and increase fruits and vegetables. It is critical to work with the family for weight loss strategies. Setting small goals each week or visit may be a useful approach and more achievable by the patient and family.

The patient should be encouraged to increase physical activity. The goal is to have moderate-

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to-vigorous exercise 60 minutes per day most days of the week. Educating the patient and parents to start slowly, with at least 10 minutes per day, is acceptable. Additionally, screen time (including television, computer, gaming systems, phone, tablets) should be limited to no more than 2 hours per day.

Because the patient is already presenting with complications due to obesity, nonpharmacologic strategies should be employed for 6 months. If the patient continues to gain weight, treatment with orlistat may be indicated. Metformin should also assist in weight management or weight loss.

5. Identify the medication-related problem and develop a solution.

The patient started risperidone approximately 6 months prior to his visit. The current dose of 8 mg daily is above the recommended 3 mg dose. The increased dose is likely contributing to the fatigue, sedation, and somnolence the patient is experiencing. Risperidone has been associated with an increased risk of developing diabetes in the first year of use. The risperidone should be discontinued, and the patient should be started on aripiprazole at 2 mg po daily for 2 days then 5 mg po daily for 2 days. The dose can continue to be titrated up by 5 mg to a maximum of 30 mg daily.

6. Devise the most pertinent patient education for the patient and caregiver.

The patient should be referred to a nutritionist for assistance with healthy food choices and to a cardiologist for an echocardiogram for evaluation of LVH. Yearly eye exams are necessary to monitor for eye disease. It is important to follow up with all of his healthcare provider's appointments.

The metformin may cause some stomach upset initially. Taking the medication with food will lessen the adverse reaction; typically, this reaction is transient and should resolve with continued use of the drug. If the patient is unable to take the drug or control his diabetes, he may have to start injections of insulin. Importance of diet and exercise as described previously should be reemphasized.

Aripiprazole has been shown to cause less weight gain than risperidone. It is important to take the medication as directed. Common adverse reactions in children include fatigue, drowsiness, and some movement disorders. The patient's mother may contact the prescriber to ask for a change from risperidone to aripiprazole due to the increased potential for weight gain and also for the increased sedative properties of risperidone as the patient has reported increase fatigue since the initiation of the current dose.

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CASE 7.2
Hypothyroidism | Level 2

Shirin Madzhidova and Sandra Benavides

1. Using Figures 1 through 3, what are the patient's current weight, height, and BMI percentiles?

Utilizing the CDC Growth Charts, this patient's weight percentile is below the 25th for his age and gender (see **Figure 4**). His height percentile is below the 5th (see **Figure 5**). The patient's calculated BMI is17.l kg/m^2. Per the CDC Growth Charts, this BMI falls at the 90th percentile (see **Figure 6**), which would categorize the patient as overweight.

2. What subjective and objective evidence supports the diagnosis of secondary hypothyroidism?

SUBJECTIVE FINDINGS: The patient presented with a chief complaint of increased fatigue; weight gain; constipation; increased difficulty with movement; gait, and balance problems; cold intolerance; and delayed growth as noted by the patient's mother.

OBJECTIVE FINDINGS: On physical exam, the patient appears to have significant growth delay, below 5th percentile for height according to his age (see Figure 5). He has a small thyroid gland on palpitation and muscle weakness of lower extremities. Laboratory findings show low free T_4 levels and thyroid-stimulating hormone (TSH) within normal range, which is consistent with findings of secondary hypothyroidism. He also has gait disturbance. Imaging studies rule out other causes of present illness, such as pituitary tumors or lesions.

3. Devise a pharmacologic regimen for the treatment of hypothyroidism that is best suited for this patient.

The recommended treatment of hypothyroidism is thyroid hormone replacement with levothyroxine due to decreased production of free T_4 by the thyroid gland. The dose of levothyroxine replacement is age and weight dependent and requires close monitoring of free T_4 levels and dose adjustments to maintain reference range levels. The recommended dose range for children 1 to 5 years old is 5 to 6 mcg/kg/day; if the initial serum T_4 is below 5 mcg/dL, treatment should begin at higher doses of 50 mcg/day. The goal serum free T_4 level is the upper third of normal range. Based on the patient's weight and age, the appropriate dosing regimen would be 88 mcg daily given in the morning on an empty stomach. Although an extemporaneous suspension can be prepared, the suspension is only stable for 8 days, which would neces-

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sitate picking up the prescription weekly from a pharmacy. Therefore, in most instances, the 88 mcg tablet can be dispensed. If administration on an empty stomach becomes a challenge, the American Thyroid Association recommends administering with the same meal daily and avoiding foods containing calcium, soy, or iron. The tablet can be crushed and mixed with 10 to 15 mL of water or apple juice.

The goal of therapy for the patient is to normalize thyroid function to avoid complications of either hypo- or hyperthyroidism. Low T_4 may decrease intellectual development and linear growth and lead to poor school performance due to impaired concentration and slowed mentation. Hyperthyroidism may adversely affect brain maturation, accelerate bone age, leading to premature closure of the epiphyses and reduced adult height. The patient may experience a period of catch-up growth that is expected with treatment. Monitoring of thyroid function tests should be done every 3 months until the patient is euthyroid and then every 6 months for monitoring and adjusting levothyroxine dose as necessary.

4. Assess the pharmacologic therapy for the patient's GH deficiency and appropriate monitoring for clinical and biochemical responses to treatment.

Panhypopituitarism is characterized by decreased production of most pituitary hormones, including growth hormone (GH), gonadotropins, and thyroid hormones. In a child with short stature or a slow growth velocity, low baseline measurements of insulin-like growth factor 1 (IGF-1), insulin-like growth factor 2 (IGF-2), and insulin-like growth factor–binding protein 3 (IGFBP-3) in the presence of deficiency of at least three pituitary hormones, suggests GH deficiency. In this patient, below-normal velocity of growth, decreased concentrations of IGFBP-3, IGF-1, TSH, and follicle-stimulating hormone (FSH), as well as delayed bone age, indicate a diagnosis of severe GH deficiency, which warrants the initiation of replacement therapy.

The goal of therapy for treatment of GH deficiency is to exogenously supplement GH to allow the patient to reach as close to normal expected adult height. Therapy includes physiologic replacement of GH using a purified polypeptide hormone of recombinant DNA origin. In children whose epiphyses are not yet fused, which is determined by radiologic examination of the hands, GH replacement usually increases growth velocity (averaging 10 to 11 cm/yr during first year of therapy). GH replacement with somatropin should be administered at a recommended dose of 0.18 mg/kg/week subcutaneously divided into equal daily doses on either 3 alternative days (0.06 mg/kg/dose) or 6 times per week (0.03 mg/kg/dose). The individualized dose for this patient would be 0.42 mg of somatropin injected subcutaneously every day, for a total weekly dose of 3 mg. Response to recombinant human growth hormone (rhGH) in pediatric patients tends to decrease over time; however, the failure to increase growth rate, particularly during the first year of therapy, indicates the need for close assessment of adherence. The injection is administered via a delivery device in an outpatient setting under the supervision of an adult caregiver. Various delivery devices are available with the various rhGH products, including automated dose delivery and needle-free devices.

The dose should be adjusted gradually based on clinical and biochemical responses assessed at monthly intervals, including body weight, height, growth velocity, waist circumference, serum IGF-1, IGFBP-3, serum glucose, lipids, thyroid function, and whole body dual-energy x-ray absorptiometry. Doses may be titrated upward if there is not an adequate response to GH therapy. An adequate response is defined as an increase in the standard deviation score (SDS) between 0.3 to 0.5, an increase in height velocity of more than 3 cm/yr in the first year, or a height velocity SDS of more than +1. Treatment should be continued until final height or epiphyseal closure or both are evident or if no response is seen at a higher dose after a 1- to 2-year trial of therapy.

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5. Devise the most pertinent patient education for the caregiver regarding levothyroxine and GH administration, monitoring, and follow-up.

Levothyroxine doses should be administered first thing in the morning on an empty stomach. Currently, no practical extemporaneous forms of levothyroxine are available for administration to pediatric patients. If the patient is unable to swallow tablets, levothyroxine may be crushed and mixed in 10 to 15 mL of water or apple juice, to be used immediately. The tablet can be given with the same meal each day if administration on an empty stomach becomes a challenge, avoiding calcium, iron, or soy-containing foods. Patients with hypopituitarism need close, ongoing, and regular follow-up by a pediatric endocrinologist, monitoring growth and free T_4 levels to assess the adequacy of T_4 replacement every 6 months to 1 year. Parents should monitor body size and symptoms and signs of T_4 deficiency (e.g., anorexia, recurrent abdominal pain, malaise) or T_4 excess (e.g., excess weight gain, Cushingoid features, hypertension).

When treatment includes rhGH therapy, instruct parents on the proper handling of the particular delivery device used and its calibration, and to recognize and report adverse reactions, such as shakiness and sweating associated with hypoglycemia or severe headache. Growth and development with GH therapy should be assessed every 3 months while monitoring IGF-1 and IGFBP-3 levels at least annually. Some potential adverse reactions may occur while taking rhGH such as local reactions at the injection site (pain, numbness, redness, and swelling), hypoglycemia, seizures, exacerbation of preexisting psoriasis, and disturbances in fluid balance. Although rare, the following complications have also been recognized: type 2 diabetes mellitus (T2DM), pseudomotor cerebri, slipped capital femoral epiphyses, scoliosis, and prepubertal gynecomastia.

6. Five days post-initiation of therapy for patient's hypothyroidism, he presents to the ED with symptoms of hypoglycemia, dehydration, and altered mental status. Upon closer evaluation, the patient was found to be in adrenal crisis. Evaluate the potential drug or disease interactions that may have precipitated the adrenal crisis and the appropriate management strategies.

Adrenal crisis occurs when there's a deficiency in circulating cortisol that can be due to impaired release from the adrenal gland. Causes for impaired release include problems with adrenal production, impaired adrenocorticotropic hormone (ACTH) secretion from the pituitary gland, impaired corticotropin release from the hypothalamus, or abruptly discontinuing exogenous corticosteroid replacement. The most likely etiology of adrenal crisis in this patient is secondary adrenal insufficiency that has resulted from pituitary disease-impairing release of ACTH. The predominant manifestations of adrenal crisis are hypotension and shock, usually with hyponatremia and hyperkalemia. Patients often have nonspecific symptoms such as anorexia, nausea, vomiting, abdominal pain, weakness, fatigue, lethargy, fever, confusion, or coma.

Due to the etiology of the patient's disease, it is important to evaluate and treat cortisol deficiency that may be present in patients with panhypopituitarism prior to initiating T_4 replacement to avoid precipitating an acute adrenal crisis. When exogenous thyroid hormone is administered, it increases the physiologic need for cortisol release as part of the negative feedback loop. However, in an impaired adrenal gland, the release of cortisol and response to ACTH is also impaired, decreasing the ability of the patient to respond to stress. The treatment for adrenal crisis is hydrocortisone 1 to 2 mg/kg/dose as an intravenous bolus, and then 150 to 250 mg/day in three to four divided doses. For this patient, the

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bolus dose would be 16 mg IV once, followed by 37.5 mg every 6 hours. Upon resolution of the acute phase, replacement doses of oral hydrocortisone at an initial dose of 10 to 15 mg/m^2/day given in three divided doses, with the first dose given as early as possible in the morning. The usual maintenance dose for children is 5 to 10 mg/dose given 3 times per day indefinitely. The American Academy of Pediatrics does not recommend the use of oral suspensions for cortisol replacement due to variable absorption. However, if the child cannot swallow tablets, a prednisone suspension can be used, remembering that it is 5 times the milligram-for-milligram potency of cortisol.

7. Identify a potential medication-related adverse reaction associated with GH replacement therapy and its management.

New onset T2DM combined with glucose intolerance and increased insulin resistance has been reported with use of GH therapy. Due to the patient's elevated body mass index and family history of T2DM, he is at increased risk for glucose intolerance or insulin resistance. The patient requires adequate patient education to improve dietary intake and increase physical activity. As he currently does not have any complications of obesity, he may not require weight loss but may require a decrease in weight gain velocity or weight maintenance. A consultation with a dietitian is warranted.

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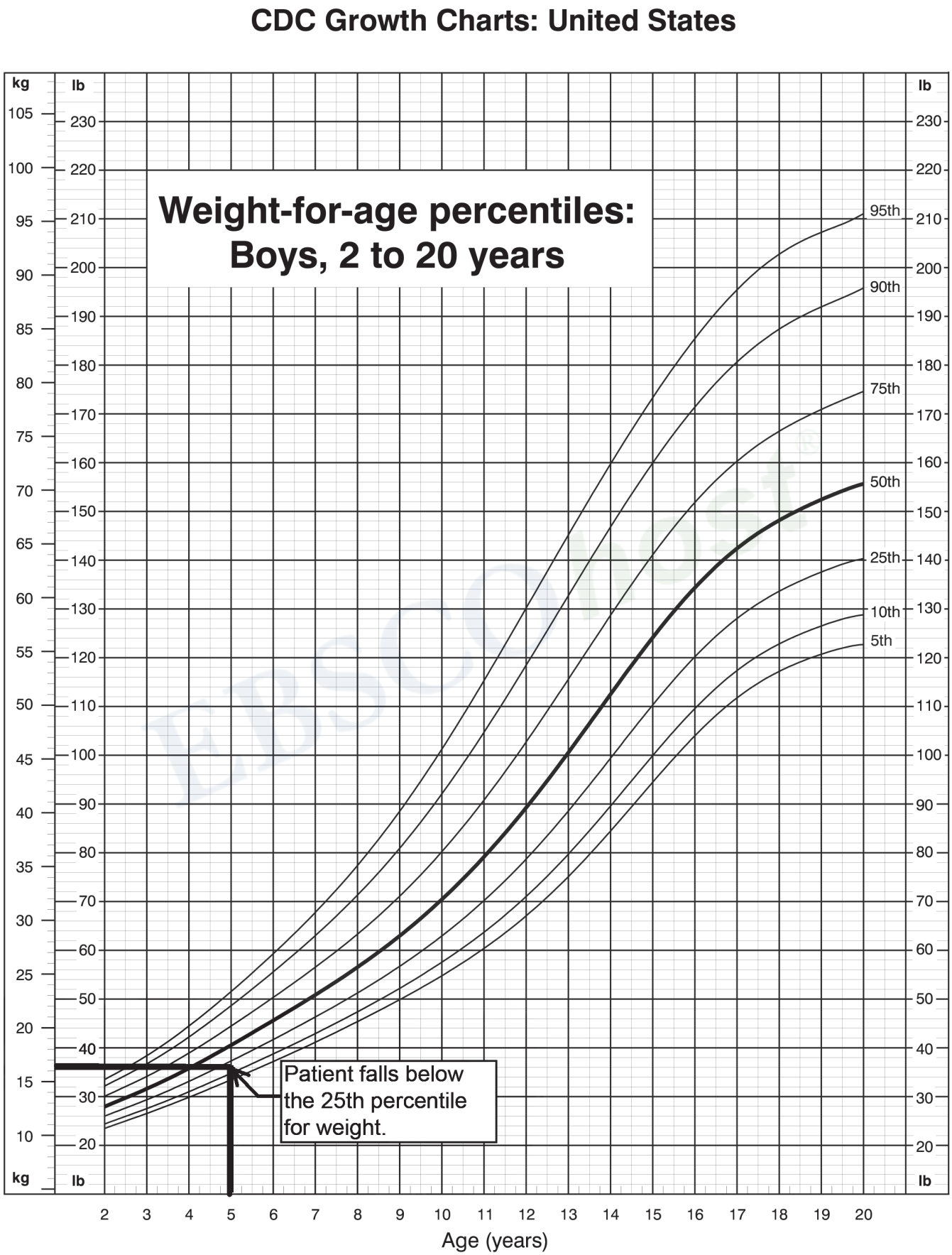

FIGURE 4: Weight-for-age percentiles, boys 2–20 years. *Source:* Kuczmarski RJ, Ogden CL, Guo SS, et al. 2000 CDC Growth Charts for the United States: methods and development. National Center for Health Statistics. *Vital Health Stat 11*. 2002;(246):1–190.

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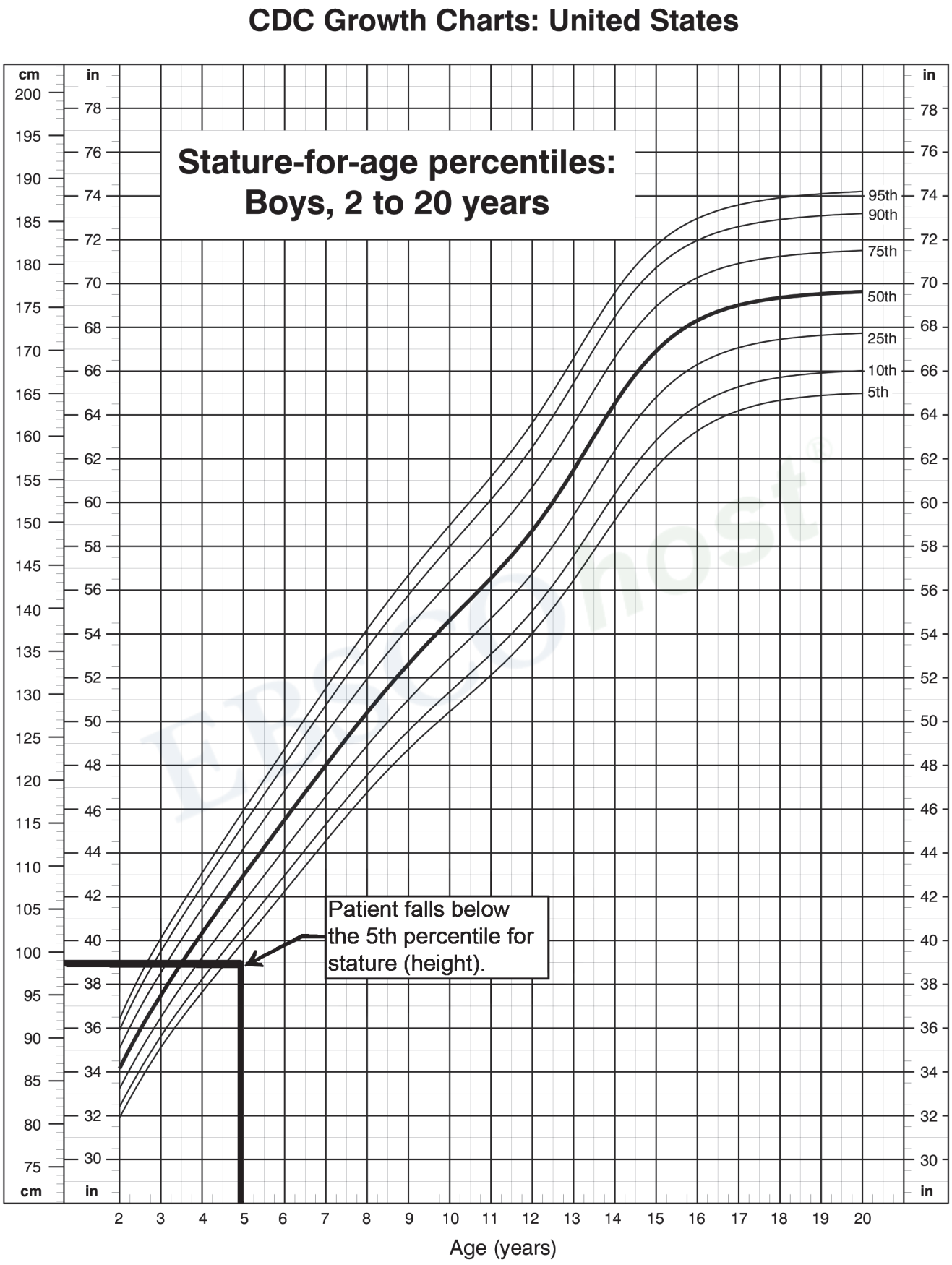

FIGURE 5: Stature-for-age percentiles, boys 2–20 years. *Source:* Kuczmarski RJ, Ogden CL, Guo SS, et al. 2000 CDC Growth Charts for the United States: methods and development. National Center for Health Statistics. *Vital Health Stat* 11. 2002;(246):1–190.

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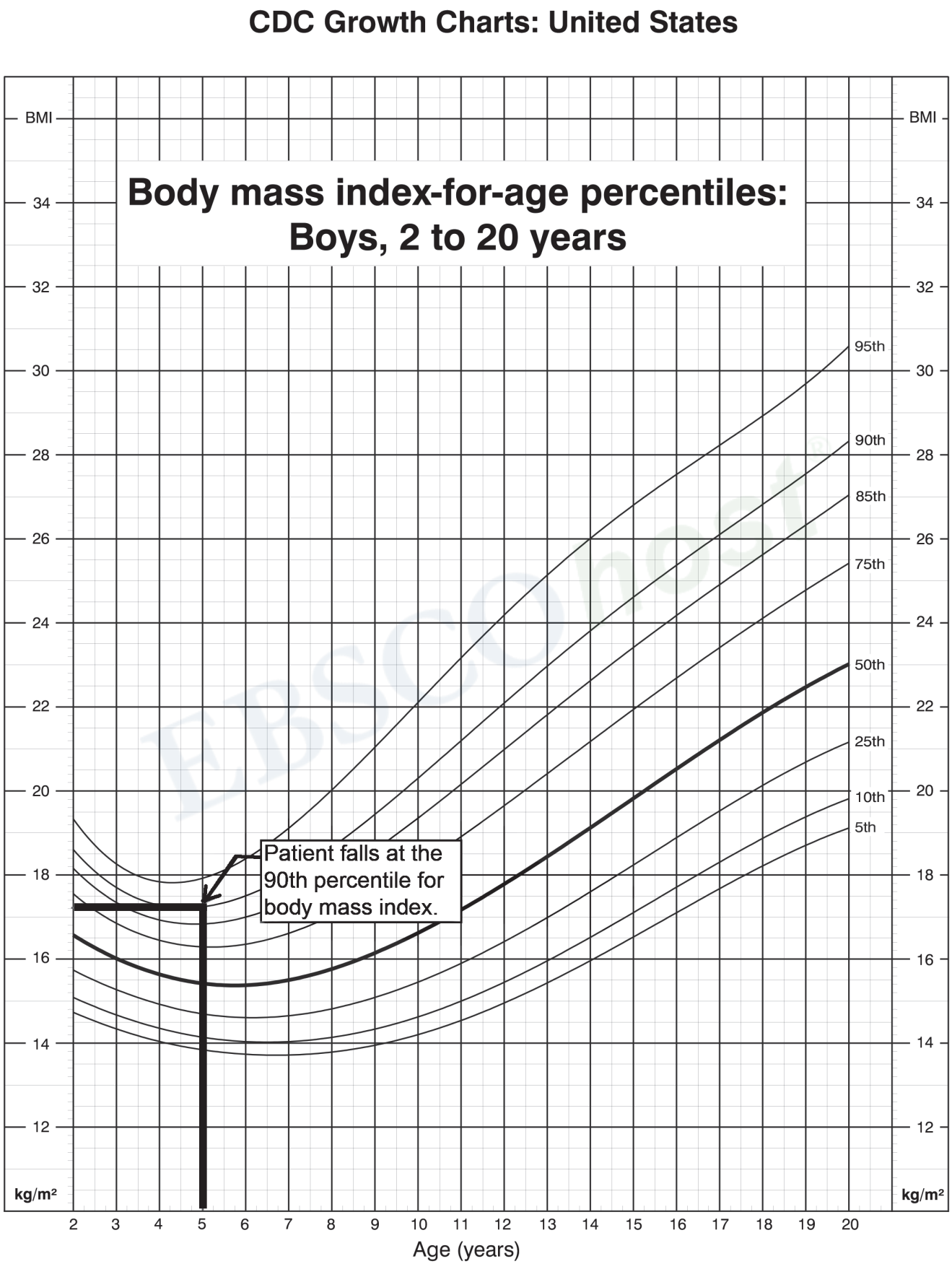

FIGURE 6: Body mass index-for-age percentiles, boys 2–20 years. *Source:* Kuczmarski RJ, Ogden CL, Guo SS, et al. 2000 CDC Growth Charts for the United States: methods and development. National Center for Health Statistics. *Vital Health Stat 11.* 2002;(246):1–190.

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CASE 7.3
Diabetic Ketoacidosis | Level 3

Julianna Crain and Sandra Benavides

1. What subjective and objective evidence supports the diagnosis of DKA in this patient?

SUBJECTIVE FINDINGS: Reported high blood glucose and ketones in urine at home, emesis, headaches, and abdominal pain

OBJECTIVE FINDINGS: Respiratory rate 40 breaths per minute with Kussmaul breathing; serum blood glucose of 647 mg/dL; glycosuria of 500 mg/dL; ketonuria greater than 80 mg/dL; metabolic acidosis demonstrated via a venous blood gas (pH= 7.14, pCO_2 = 25 mm Hg, HCO_3 = 9.8 mmol/L, BE = –18.8 mmol/L, SpO_2 = 86.1%); anion gap = 37 mmol/L; Cl^- = 96 mmol/L; and GCS = 15

Based on the patient's presenting laboratory values, she is considered to have moderate DKA with a venous pH less than 7.2 and the HCO_3 less than 10 mmol/L. The likely precipitating event for DKA in this patient was the acute illness and nonadherence to insulin therapy.

2. Describe the necessary laboratory parameters to obtain before developing an appropriate pharmacologic regimen for this patient.

When a patient arrives to the ED with DKA, cardiac monitoring (e.g., ECG) and pulse oximetry are imperative to assess for arrhythmias secondary to hyperkalemia and tissue oxygenation, respectively. Throughout the acute phase, monitoring vital signs—including BP, HR, RR, SpO_2, temperature, intake/output, and GCS—are essential because patients are at greater risk for cerebral edema during this time. A venous blood gas, urinalysis, HbA_{1c}, and urine pregnancy test in females should be done on admission to the ED. In addition, bedside blood glucose monitoring must be done every hour, and a comprehensive metabolic panel (CMP)—including phosphorous and magnesium—are monitored approximately every 4 hours during the acute phase. Depending on the patient's status, this length of time could range from 24 to 48 hours.

Cerebral edema is a complication of DKA with the potential to cause irreversible damage. If a patient has symptoms of cerebral edema, namely altered mental status, a prompt reduction in intracranial pressure is required with the administration of mannitol (dosed at 0.25 to 1 g/kg/dose) or 3% hypertonic saline (dosed as a continuous IV infusion at 0.1 to 1 mL/kg/hr). The onset and progression of cerebral edema usually develops within the first 4 to 12 hours after treatment

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initiation; however, it may occur prior to treatment or develop as late as 24 to 48 hours after treatment. Therefore, monitoring should occur hourly in the first 4 to 12 hours and every 4 hours for the next 1 to 2 days after treatment initiation.

3. What are the overall goals of therapy related to DKA? Given these goals, develop a pharmacologic regimen for the treatment of DKA.

There are three overall goals related to the treatment of DKA. Patients presenting with DKA are often dehydrated due to the osmotic diuresis induced by glycosuria. Therefore, the first goal is to provide fluid replacement and volume expansion to improve oxygenation and tissue perfusion. This is necessary to prevent multisystem organ failure. On presentation of a patient with DKA, hydration status must be assessed. The patient was dehydrated as indicated by BUN = 39 mg/dL and SCr = 2.3 mg/dL. Her physical exam had positive findings of hypoxemia (SpO_2 of 86%), hypotension, tachycardia, and tachypnea, which collectively indicate dehydration and volume depletion. IVFs are necessary to expand the intravascular space and improve tissue perfusion and oxygenation. The patient received an IV isotonic crystalloid 0.9% saline at 20 mL/kg over 1 to 2 hours. For this patient, the dose of 700 mL IV over 1 hour was appropriate. The repeat bolus dose was also appropriate if the patient remained hemodynamically unstable indicated by the continued presence of hypotension, tachypnea, and tachycardia. Resuscitation volumes should never exceed 40 to 50 mL/kg (1,750 mL for this patient) in the first 4 hours to avoid the risk of cerebral edema and herniation.

The second goal of therapy is to suppress ketone production by stopping lipolysis (breakdown of triglycerides into free fatty acids and glycerol) and ketogenesis (production of ketone bodies by the liver as an energy source for the brain and heart in periods of starvation). This is accomplished with the administration of insulin, which will also decrease the elevated glucose. In this patient, 1 to 2 hours following the initial fluid bolus, an insulin continuous infusion was started to halt the production of ketones and reduce serum glucose concentrations. The insulin dose of 0.1 units/kg/hr (3.5 mL/hr) in normal saline was appropriate and should be continued until the ketosis resolves, which is indicated by a pH greater than 7.3, bicarbonate level greater than 15 mmol/L, or a resolved anion gap. Reducing the serum glucose level must not exceed a rate of 100 mg/dL/hr; if this rate is exceeded, the insulin dose must be decreased to 0.05 units/kg/hr. The principal outcome and primary goal of insulin therapy is to suppress lipolysis and to reverse ketosis more so than to decrease the serum glucose concentration. Once the ketone production has stopped and oral intake is tolerable, the IV insulin infusions may be transitioned to the subcutaneous (SC) route. The IV infusion should be discontinued 2 hours after the patient eats and SC insulin is administered. At that point, the patient can resume her home insulin pump settings.

Lastly, the third goal of therapy is to restore electrolyte balance and avoid complications such as hypokalemia, hypophosphatemia, hypoglycemia, or cerebral edema. The devastating complication of DKA is cerebral edema; therefore, appropriate management of DKA is crucial. After the initial IVF saline bolus, the remaining fluid deficit should be administered over the next 48 hours with either 0.45% or 0.9% saline infusion depending on the patient's sodium level. Because of the low initial sodium level of 130 mmol/L, the most appropriate option for this patient is 1 liter of 0.9% saline, which contains a higher amount of sodium (154 mEq/L) than the 0.45% saline. The rate of infusion for the IVF should include the maintenance rate plus additional necessary fluid replacement depending on the degree of dehydration on presentation but should not exceed twice the maintenance rate (in this case, 75 mL/hr). The maintenance rate is calculated utilizing the Holliday-Segar method. The patient weighs approximately 35 kg, which would be 1,500 mL for the first 20 kg and 20 mL/kg for each kilogram greater than 20 kg (20 mL/kg × 15 = 300 mL) or 1,800 mL/day. Because this patient initially presented dehydrated, the replacement fluid in normal saline should be infused at a rate of 112.5 mL/hr (approximately 1.5 times the maintenance rate).

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In the initial stages of DKA, patients often have elevated serum potassium levels due to insulin deficiency and the shift of potassium extracellularly; therefore, the addition of potassium to the initial saline infusion is often unnecessary. This patient's potassium level at arrival was 5.7 mmol/dL, and initial IVFs did not include potassium. The potassium levels may decrease and normalize as insulin is administered; therefore, the addition of potassium at a concentration of 40 mEq/L in the IVF is indicated when the insulin infusion begins to prevent hypokalemia. However, it is also necessary to wait to initiate the potassium until the patient has voided to avoid hyperkalemia.

Transitioning to dextrose (D5W) in place of the normal saline in the IVF occurs once the serum glucose concentration approaches 250 to 300 mg/dL in order to prevent subsequent hypoglycemia. At this time, this patient's IVF should be changed to 1 liter of D5W and 40 mEq of potassium as a continuous infusion. The rate of infusion varies and should be administered either at maintenance or 1.5 times the maintenance rate (75 to 112.5 mL/hr), depending on the patient's hemodynamic instability and labs (e.g., sodium, potassium, and glucose concentrations). This provides the patient with approximately 0.1 mEq/kg/hr of potassium and 0.1 g/kg/hr of dextrose. Of note, generally if the serum glucose concentration falls below 150 mg/dL, the dextrose in the IVF should again be changed to D10W and insulin infusion rate reduced to 0.05 units/kg/hr to avoid hypoglycemia.

Phosphate is lost with osmotic diuresis during DKA. IVFs as well as insulin will decrease phosphate levels. Evaluation of the patient's serum phosphate level and symptoms of hypophosphatemia, such as muscle weakness or decreased cardiac function, are necessary as a consideration for phosphate replacement. Phosphate can be replaced by utilizing a potassium formulation, such as potassium phosphate, in the IVF or as single replacement doses. This patient did not require phosphate supplementation or replacement as there is no evidence of hypophosphatemia and the CMP indicates a slightly elevated serum phosphate level. There is a lack of evidence for magnesium replenishment during DKA because serum magnesium levels will normalize as acidosis is corrected.

Sodium bicarbonate replacement is not recommended or indicated in managing acidosis during DKA because administration increases the risk for cerebral edema.

4. Describe appropriate management of type 1 diabetes mellitus during an illness and potential strategies to avoid DKA in the future.

During an illness, it is important for the caregiver and the patient to continue to administer insulin. The basal insulin dose (or less) may be sufficient to prevent the development of DKA. The patient may be advised to administer 5% to 10% of the total daily dose every 3 to 4 hours. If the patient cannot eat, advise the patient to drink a sugar-containing beverage or snack. If the patient is unable to tolerate a sugar-containing beverage or snack, advise the patient to administer glucagon at 10 mcg per year of age (140 mcg) to prevent hypoglycemia. The patient should be warned that the glucagon may worsen her nausea and vomiting, and for that reason the aforementioned dose is recommended to minimize adverse reactions.

Equally important is the need for self-monitoring of blood glucose and monitoring of urine for the presence of ketones during an illness. The blood glucose should be monitored every 3 hours, and the urine ketones should be checked at least twice a day. If ketones are present in the urine, the insulin dose may have to be increased to 10% to 20% (from the 5% to 15%) of the total daily dose every 3 to 4 hours to prevent DKA. Patient-specific doses and guidelines should be provided to the family.

Lastly, advise the patient to remain hydrated, if possible, when experiencing an illness. This will also aid in the prevention of DKA. Close communication with a healthcare provider is also warranted.

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5. Describe the importance of immunizations in this patient.

Diabetes weakens the immune system and places the patient at risk for a severe influenza infection as well as hospitalization. Also, this patient is at risk for developing secondary bacterial infections, such as pneumonia. The Centers for Disease Control and Prevention (CDC) recommends all patients with diabetes to receive both the pneumococcal and annual seasonal influenza vaccinations. For this patient, she should receive the 23-valent polysaccharide vaccine (PPSV23—Pneumovax®) if she has not yet received it. The patient should also receive the inactivated influenza vaccine yearly. It is not advised to administer the live, intranasal vaccine to patients with diabetes.

6. Identify and resolve the medication-related problem(s).

The nonadherence to insulin contributed, in part, to the onset of DKA. During an illness, insulin requirements may increase despite limited food intake due to increased counter-regulatory hormone secretion (e.g., catecholamines, cortisol, growth hormone). These hormones oppose the effects of insulin and can increase glucose levels resulting from gluconeogenesis and glycogenolysis. During a time of illness, it is important for the caregiver to be in communication with a healthcare provider or follow a predetermined protocol to monitor and adjust insulin levels as mentioned above.

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CASE 8.1
Attention Deficit Hyperactivity Disorder | Level 1

Jennifer E. Thomas and Joshua Caballero

1. What is the subjective and objective evidence for the diagnosis of ADHD and ODD?

SUBJECTIVE FINDINGS: ADHD—difficulty concentrating at school, frequent careless errors at school, inability to concentrate when spoken to, and trouble completing tasks at home; ODD—anger outbursts and history of starting fights at school and at home

OBJECTIVE FINDINGS: There is no objective evidence to support the diagnosis of ADHD and ODD in this patient. Although ideally one would like scales available to determine efficacy for ADHD (e.g., Conners' Rating Scale, Vanderbilt ADHD Rating Scale) and ODD (e.g., subscales or specific questions from aforementioned scales), they are not always available in clinical practice.

2. What risk and environmental factors may have contributed to ADHD in this patient?

Low birth weight and possible early social deprivation are potential risk factors for the development of ADHD in this patient as his mother had postpartum depression. There is evidence that there may be a genetic component to ADHD. The father appears to display symptoms of ADHD given his history of not completing tasks.

3. How would you assess the current pharmacologic therapy for the patient?

Current therapy is not working and possibly contributing to insomnia. The patient has not experienced an improvement in concentration or disruptive behavior at school. Therefore, the patient requires a dose reduction (to alleviate the insomnia) and the addition of an alternative agent (to treat the unresponsive ADHD symptoms and aggressive behaviors). In this case, the increase in dose led to insomnia, necessitating a decrease in the dose. When this occurs, it is best to reduce the stimulant to the previous dose and add another agent to treat the unresolved symptoms of ADHD and aggression.

In this case, the patient is overweight (BMI greater than 90th percentile) and has an elevated blood pressure (prehypertension as blood pressure is above the 90th percentile) and heart rate; he may benefit from an alpha-2 agonist. With the addition of an alpha-2 agonist, a reduction of the previous stimulant dose may be necessary. For example, studies with clonidine indicated a 15% to 40% decrease in

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stimulant doses when clonidine was titrated to effect. Therefore, after one decreases amphetamine/dextroamphetamine XR back to 25 mg daily, further reduction *may* be needed after initiating an alpha-2 agonist. However, this further reduction in stimulant dose would not occur until the alpha-2 agonist is at a therapeutic dose and patient is reassessed.

4. What pharmacologic treatment would you recommend?

Although amphetamines are first-line therapy for the treatment of ADHD, this patient was not able to tolerate an increased amphetamine dose. Therefore, the amphetamine should be reduced to its previous dose (in this case 25 mg daily), which was partly effective. The patient will also require an adjunctive agent for the management of his symptoms.

Start guanfacine extended-release (Intuniv®) 1 mg at bedtime. Titrate by 1 mg at weekly intervals up to 4 mg at bedtime. Although there are many other adjunctive treatments to use, alpha-2 agonists may be preferred in those with ODD, as they are effective in reducing aggression and oppositional symptoms. Unlike other treatment options, alpha-2 agonists may also have a beneficial effect on the patient's blood pressure, which is elevated.

Among the alpha-2 agonists there are two options: guanfacine and clonidine (see **Table 1**). Guanfacine has a couple of advantages over clonidine. Guanfacine can be dosed once daily and has a perceived decreased risk of adverse reactions, due to its higher selectivity for alpha-2 receptors. Guanfacine is available in both an immediate-release and an extended-release formulation. The extended-release formulation may be preferred in this case because the patient displays ODD symptoms throughout the day. Also, once-daily dosing may help with adherence. However, please note that if cost is an issue, the immediate-release formulation can be used.

5. How would you counsel the patient and caregiver regarding the new medication?

Recent studies state that approximately 20% of patients discontinue their ADHD medication within 1 year. From these, over 40% of patients discontinue during the first month largely due to lack of efficacy and the occurrence/possibility of adverse reactions. As such, it is important to stress that alpha-2 agonists take approximately 2 to 4 weeks before benefits are seen. Also, efficacy is usually assumed by a 30% to 50% reduction in symptoms. Therefore, expectations and goals of therapy should be discussed with the patient and family. Goals of therapy for this patient include improved concentration span and a reduction in aggression, while minimizing adverse reactions from the medications. Guanfacine may cause drowsiness and dizziness and, therefore, should be taken at bedtime. The package insert states one can start in the morning; however, reports of sedation by patients occur at high rates (~38%). The patient should let the prescriber know immediately if the medication causes excess dizziness, bradycardia, or syncope (from hypotension). Patients should make sure to drink plenty of fluids to prevent dehydration, which would increase the risk of dizziness. Additionally, it is best to separate the extended-release formulations of guanfacine from high-fat meals, which increase absorption of the medication. Alpha-2 agonists may cause dryness of the mouth. If this occurs, the patient can try using sugarless gum or sugarless hard candy (e.g., xylitol lollipop). Patients should not stop the medication abruptly unless they are at the starting dose or a severe adverse reaction occurs. Discontinuing without

TABLE 1: BENEFITS AND CONSIDERATION OF ALPHA-2 AGONISTS

	Pros	Cons
Clonidine	Absorption not affected by food Available in weekly patch formulation	Extended-release formulations still require twice daily administration
Guanfacine	Higher selectivity for alpha-2 receptors, which may result in a decreased incidence of adverse reactions Can be dosed once daily	Extended-release formulation should be separated from high-fat meals

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tapering can lead to withdrawal syndrome (e.g., rebound hypertension, headache, tremor, restlessness, nausea). However, some suggest the withdrawal is less with guanfacine (3%) versus clonidine (50% to 80%).

6. The patient returns for follow-up after 8 weeks. At this point, the guanfacine extended-release dose has been increased to 2 mg at bedtime. The patient's parents state that he has been feeling very dizzy, and he fainted yesterday. The patient's blood pressure is 90/50. Upon questioning the parents about how the medication is administered, they state that he usually eats dinner around 8 p.m. and takes his guanfacine dose after finishing his meal with dessert. What could be the cause of the adverse reaction? How will you instruct the patient to proceed?

When taken with a high-fat meal, guanfacine extended release can have an increase in AUC of approximately 40% and an increased C_{max} of approximately 75%. The patient may be experiencing an adverse reaction due to increased absorption of the guanfacine. Advise the parents to avoid high-fat meals in the evening, to try to have the child eat earlier, and to allow more time between dinner and his guanfacine dose. An overall lower-fat diet should also be encouraged for this patient, considering he is overweight. Alternatively, if this is not possible because the routine has been set and the patient will be more adherent with the medication with dessert, then the guanfacine dose will need to be decreased. The dose should be decreased 1 mg at bedtime if the patient prefers to take the medication with dessert. Another option would be to switch to a morning dose; however, the change may cause an increase in adverse reactions (e.g., sedation). The patient should be instructed to follow up in 4 weeks after making changes in the dose to assess for efficacy and adverse reactions. If the patient continues experiencing dizziness, the prescriber should be contacted.

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CASE 8.2
Autism Spectrum Disorder | Level 2

Jose Valdes and Jose A. Rey

1. What subjective and objective evidence supports the diagnosis of ASD?

Identified risk factors for ASD (not present in this patient) are closer spacing of pregnancies; advanced maternal or paternal age (generally ages greater than 35 years old); and extremely premature birth (less than 26 weeks gestational age).

SUBJECTIVE FINDINGS: There is a delay in or total lack of the development of spoken language—the content of her speech is comparable to that of a 24-month old. She has marked impairment in the ability to initiate or sustain a conversation with others. There is an inflexible adherence to specific nonfunctional routines or rituals, e.g., mother states, "She insists on having me tie her shoes left to right before breakfast, otherwise she will hit me!" She is more socially withdrawn from other children and has had difficulty developing peer relationships appropriate to her developmental level. There is a lack of social or emotional reciprocity; she maintains a flat affect during social interactions. She exhibits a persistent preoccupation with parts of objects, e.g., focusing on the wheels of a toy bus in the classroom.

OBJECTIVE FINDINGS: The findings on physical exam are agitation, bruising around eyes from self-injurious behavior, tachycardia, respiratory depression, and fine hand tremor.

She has a childhood autism rating scale (CARS) score of 38. Using the CARS, the cutoff for diagnosis of ASD is at least 30 for mild ASD. Scores of 30 to 37 indicate mild-to-moderate ASD and scores of 38 to 60 indicate severe ASD. Scores range from 15 to 60 for the CARS. Average sensitivity and specificity for the CARS were 89% and 82%, respectively, for autism; and 82% and 80% for ASD.

2. Assess the nonpharmacologic and pharmacologic therapy for ASD and seizure disorder.

ASD: The patient did not present with pharmacologic treatment, which would be appropriate if the patient did not present with symptoms prior to the episodes and was controlled on behavioral techniques. Pharmacologic treatment should only be considered when there is a specific target symptom (hyperactivity, inattention, impulsivity, aggression, self-injury, anxiety, obsessive-compulsive behaviors, rigidity, repetitive behaviors, and depressive symptoms) or coexisting

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condition. She is currently on a casein-free and gluten-free diet. Cochrane Collaboration review of gluten-free and casein-free diets in ASDs showed significant improvements in overall autistic traits, social isolation, communication, and interaction. The report noted there were no harmful outcomes. If the parents and child feel there has been improvement on this diet, it may be continued.

MEDICATION-RELATED PROBLEM: The patient is currently presenting with signs of acute valproic acid toxicity. Her valproic acid level is 139 mcg/mL, and the symptoms she presents with are hypotension, respiratory depression, tachycardia, tremor, borderline serum ammonia levels, and agitation. However, the mother said the agitation was related to her not getting her treats. The patient is currently on 1,000 mg/day, calculated to be approximately 57 mg/kg/day. Most patients' seizures are controlled at doses below 60 mg/kg/day; however, given her symptoms and valproic acid level, she appears to be experiencing toxicity.

- Supportive care is the principal treatment for valproic acid toxicity and results in positive outcomes for the vast majority of patients. This involves management of fluid status, autonomic system control, and hemodynamic support. This would be optimally managed in the inpatient hospital setting.
- Carnitine supplementation has been shown to reduce symptoms in patients with valproic acid-induced hyperammonemia and hepatotoxicity. This may be caused in part by a carnitine deficiency to which carnitine supplementation may prevent and attenuate these adverse reactions. In this case, carnitine supplementation would not be necessary as the patient does not present with lethargy, elevated ammonia levels, or with hepatic dysfunction. However, there are no adverse reactions related to carnitine therapy for valproic acid toxicity so in the absence of clinical or laboratory signs of toxicity, oral carnitine can be administered prophylactically with a dose of 150 to 500 mg/kg per day (up to 3 g/day) divided every 6 hours given by mouth. The patient weighs 17.6 kg; thus, the appropriate dose can be rounded to 2,640 mg (8,800 mg divided every 6 hours). However, with a maximum dose at 3,000 mg (3 g), 750 mg by mouth every 6 hours would meet that maximum dose.
- Gastrointestinal decontamination would not be necessary because the toxicity was a result of chronic administration of the medication as opposed to an acute overdose.

SEIZURE DISORDER (PHARMACOLOGIC): Valproic acid requires therapeutic drug monitoring and has many drug interactions similar to other older anticonvulsants (e.g., carbamazepine, phenobarbital, phenytoin). Although the patient's seizures were controlled on this medication and valproic acid was an appropriate agent for the patient, some may be hesitant to restart this medication provided that the patient had toxicity with it. If chosen to continue this medication, hold valproic acid therapy and monitor the patient's serum level daily. Restart valproic acid at a low dose of 10 to 15 mg/kg/day (176 to 264 mg/day) once serum levels fall within normal range. A dose of 200 to 250 mg can be administered easily in the liquid formulation as doses of 4 mL or 5 mL, respectively, from the 250 mg/5 mL stock bottle. Serum levels may be monitored weekly after the patient has restarted therapy to aid in adjusting the dose to a therapeutic level. Also seizure control, liver function tests, cognitive impairment, vital signs, and neurological status should all be evaluated monthly until steady-state has been reached and may advance to every 3 to 6 months thereafter. The goal is to continue to keep the patient seizure free. As the half-life of valproic acid is 7 to 13 hours, the serum levels would be expected to drop over 2 to 3 days. Once the patient is restarted on valproic acid, the dose may be increased by 5 to 10 mg/kg/day at weekly intervals until therapeutic levels are achieved.

3. Develop an alternative plan to treat the patient's seizure disorder.

Lamotrigine, oxcarbazepine, and topiramate are three newer anticonvulsants with a more favorable drug profile than valproic acid and

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can act as suitable alternatives for this patient if the family chooses. If a new agent is chosen, valproic acid should be tapered off after starting the new agent by decreasing the total dose weekly by 20% to 25% unless otherwise specified. Oxcarbazepine would be the first choice as it has a favorable adverse affect profile, ease of dosing, and lack of therapeutic drug monitoring. Lamotrigine would be chosen second as topiramate may cause unwanted adverse reactions, such as weight loss and changes in mood.

- ❑ Oxcarbazepine (Trileptal®)
  - Initiate at 8 to 10 mg/kg/day po given in two divided doses (75 mg po bid in this patient).
  - Dose can be titrated upward by 5 mg/kg/day every third day to the recommended daily maintenance dose of 600 to 900 mg/day po.
  - Taper valproic acid over a 3- to 6-week period while increasing the oxcarbazepine dosage in increments no greater than 10 mg/kg/day at weekly intervals to achieve the recommended daily monotherapy dose. Laboratory monitoring of oxcarbazepine is not necessary, only monitoring for drug efficacy and adverse reactions every 2 to 4 weeks initially, then 4 to 8 weeks once the patient is at the recommended maintenance dose. The goal is to continue to prevent seizures from occurring as the patient has been seizure free since starting therapy with valproic acid.
  - Adverse reactions include hypotension, lower extremity edema, confusion, fatigue, headache, insomnia, skin rash, hyponatremia, weakness, and dry mouth.

Nonpharmacologic therapy includes the initiation of a ketogenic diet, which may help to reduce the risk of seizure activity. Because the patient has been controlled on pharmacotherapy, initiating the patient on a ketogenic diet is not necessary. It consists of a diet high in fats and low in protein/carbohydrates. One study estimates a complete cessation of all seizures occurring in 16% of patients, a 90% reduction occurring in 32% of patients, and greater than 50% reduction of seizures occurring in 56% of patients. Another study revealed that parents of children with ASD perceived a ketogenic diet to contribute to the improvement of seizures and other clinical factors.

Initiation of the ketogenic diet may necessitate hospitalization at first as hypoglycemia, dehydration, and vomiting may occur. Patients are monitored at baseline, then every 3 to 6 months thereafter. Long-term adverse reactions could include an increase in hepatic and pancreatic enzymes, weight loss, hypoproteinemia, nephrolithiasis, constipation, and hyperlipidemia. Vitamin supplementation will be necessary because patients maintained on the ketogenic diet are at high risk of nutritional deficiency. Reports of thiamine, vitamin D, vitamin C, and selenium deficiencies have been found in children.

Patients on a ketogenic diet should not be initiated on valproic acid due to the risk of hepatotoxicity from the diet and valproic acid. If a ketogenic diet is started, patients may benefit from alternative anticonvulsant treatment such as lamotrigine, oxcarbazepine, or topiramate. Other diets such as the modified Atkins or low glycemic index diet (LGID) may be effective alternatives. Preliminary studies have shown that the modified Atkins diet can help some children with intractable seizures. Data for the modified Atkins diet or the LGID are not as robust compared to the ketogenic diet. It is recommended to consult a neurologist before initiating a diet.

4. Discuss treatment outcomes and goals to using pharmacotherapy for reducing target symptoms of ASD as well as pharmacotherapy for the seizure disorder.

For ASD, a clinically meaningful reduction in problematic and maladaptive behaviors (e.g., agitation, aggression, self-injurious behavior) is the desired outcome so that individuals can then engage in the nonpharmacologic treatments designed to improve their overall functional status and impaired domains. A goal of therapy should be to target specific symptoms the patient is having (e.g., agitation, aggressiveness, and self-injurious behavior) that inter-

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feres with the individual's ability to participate in other treatments designed to improve functional ability. By reducing those target symptoms, patients are able to engage occupational or speech-language therapists, psychologists, etc., and can continue making progress through nonpharmacological means by improving their functional impairments.

Therapeutic outcomes for patients with seizure disorder should result in a decrease in the frequency of seizures, with the goal of complete seizure resolution. Patients should also experience an improved quality of life and be free of adverse reactions that may be caused by pharmacologic treatment. Withdrawal of anticonvulsant therapy can be considered if a patient has been absent of seizures for 2 to 5 years while on pharmacologic therapy, has a single type of partial or primary generalized tonic-clonic seizure, has a normal EEG with pharmacologic treatment, and has a normal IQ/neurological examination.

5. Develop a pharmacotherapeutic regimen for the treatment of the acute agitation/aggression and self-injurious behavior and discuss points to monitor.

Pharmacologic treatment strategies are designed to target an aspect of a patient's disorder that interferes with participation in other treatments that are designed to achieve maximum functional ability. In this case, the patient is agitated and as a result becomes aggressive and self-injurious as well as unable to participate in speech and occupational therapy. Currently, two agents are FDA approved for irritability associated with ASD; however, both agents have warnings for lowering seizure threshold, as do all antipsychotics. The patient is being monitored and treated for seizures so therapy should begin at low doses and titrated as needed.

- ❑ Risperidone (Risperdal®)
  - Initially, administer 0.25 mg po daily. After a minimum of 4 days, the dose may be increased to the target dose of 0.5 mg po daily. Maintain target dose for at least 14 days. Thereafter, increase dose as clinically necessary at intervals of at least 2 weeks and increments of 0.25 mg/day po. The effective dose range is 0.5 to 3 mg/day. In patients weighing less than 20 kg, the maximum therapeutic effect is typically reached at 1 mg/day.
  - Monitor agitation, irritability, weight, and blood glucose every month. Lipids, serum prolactin, and liver function can be assessed every 6 to 12 months. Cardiac function (ECG) can be assessed at baseline prior to starting risperidone and yearly thereafter.
  - Adverse reactions include somnolence, fatigue, increased appetite, tremor, hypersalivation, extrapyramidal symptoms (EPS), and weight gain.
- ❑ Aripiprazole (Abilify®)
  - Initially, administer 2 mg/day po. Increase dose to 5 mg/day after 1 week. The dose may be increased to 10 to 15 mg/day thereafter if needed. Dose changes should occur in increments of 5 mg/day at intervals of no less than 1 week. The recommended dose is 5 to 10 mg/day. The maximum recommended dose is 15 mg/day.
  - Aripiprazole has an FDA indication for children greater than 6 years of age. Limited data are available for children less than 6 years of age.
  - Monitor agitation, weight, and blood glucose every month. Lipids and liver function can be assessed every 6 to 12 months.
  - Adverse reactions include sedation, insomnia, fatigue, vomiting, tremor, weight gain, hypersalivation, decreased appetite, and EPS.

The patient will benefit from a trial of risperidone. If the patient does not achieve improvement once the dose is titrated to 1 mg/day, the dose can be increased (if tolerated), or the medication can be switched from risperidone to aripiprazole.

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6. What information would you provide to the caregivers and child to help manage symptoms of newly prescribed medications?

- ❑ Counsel patients and caregivers on the adverse reactions mentioned above for all new medications prescribed.
- ❑ Provide education on what to expect in regard to the patients' resolution of symptoms. Pharmacotherapy in ASD is for symptomatic management only. Although improvements in the core features of ASD (impairments in social interaction or reciprocity, communication and language, and repetitive or stereotypic behaviors) could be made through other behavioral or educational/occupational interventions, the need to maintain pharmacotherapy for symptoms should be reassessed periodically.
- ❑ Provide structure with regard to daily activities for children with ASD. Consistency, schedules, and rewards for good behaviors are important for children. Also provide safety for patients as they learn to adapt to the new medications, keep dangerous objects out of reach or out of the home, set boundaries, and create an area where children can feel relaxed and safe. Pay special attention to children, and report any adverse reactions or differences in behaviors to their physicians.
- ❑ Be consistent in dosing schedules and appointments with physicians. This will aid in preventing missed doses and allow for children to be seen regularly to track their progress.

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CASE 8.3
Depression | Level 2

Kristen Lamberjack and Christine Prusa

1. What subjective and objective evidence supports the diagnosis of pediatric depression?

SUBJECTIVE FINDINGS: The patient presented with a chief complaint of depression, which is improving on her current therapy. She described symptoms of depressed mood (sadness) most days, anhedonia (inability to experience pleasure from activities usually found enjoyable), irritability, lack of motivation (she missed 50 days of school), low self-esteem, appetite changes with weight loss, guilt, sleep disturbances, and crying episodes. To meet the diagnosis for a major depressive episode, patients must have five of nine depressive symptoms for 2 consecutive weeks with one of those symptoms being depressed mood or anhedonia.

OBJECTIVE FINDINGS: Thyroid function tests rule out thyroid disorder as a diagnosis for her symptoms of fatigue and sleep disturbance. Her normal hemoglobin and hematocrit levels also help to eliminate anemia as a likely cause for her low energy levels. Physical exam displays moderate anxiety and nervousness.

She also has a family history of depression (mother, maternal grandmother, and aunt).

2. Assess the pharmacologic therapy for the patient's depression treatment and provide a treatment plan to optimize therapy.

The patient is responding to therapy with fluoxetine; however, she is not yet in remission of symptoms. The fluoxetine dose was increased at her last visit by the PCP, and she is now experiencing adverse reactions from her therapy. Anxiety and nervousness is a common adverse reaction of fluoxetine with a rate of 3% to 15%. The patient is responding to therapy with a selective serotonin reuptake inhibitor (SSRI); therefore, keeping her on a medication in the same class would be the best course of action.

Depending on the length of time a patient is taking an SSRI, he or she may have withdrawal symptoms when stopping therapy. When switching SSRIs, patients may have to cross taper (decrease one while increasing the other) therapy; however, fluoxetine has a long half-life and the patient is taking ≤20 mg; therefore, when changing therapy from fluoxetine to another SSRI, the patient should stop the fluoxetine 20 mg capsules and wait 4 to 7 days before starting the new SSRI at the initial dose.

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The best option in this patient would be escitalopram 5 mg daily, increasing to an effective dose of 10 to 20 mg and a maximum dose of 20 mg. The dose can be increased every 4 to 6 weeks to effective dose based on response to therapy and if the patient experiences no intolerable adverse reactions. Escitalopram is the only other FDA-approved antidepressant in patients 12 and older for major depressive disorder (MDD) other than fluoxetine, and it is less likely to cause anxiety and nervousness. It is also recommended as first line after fluoxetine for patients 12 and older according to the Guidelines for Adolescent Depression in Primary Care (GLAD-PC).

Other potential alternatives that would be considered second line are sertraline 25 mg daily, increasing to an effective dose of 100 mg with a maximum dose of 200 mg or citalopram 10 mg daily, increasing to an effective dose of 20 mg with a maximum dose of 60 mg. Although not FDA-approved in pediatrics, both medications have been studied in pediatrics with evidence supporting their use. They are also less likely than fluoxetine to cause anxiety and nervousness. Paroxetine has the least data supporting its use in pediatrics and is more likely to cause adverse reactions and would not be considered a potential option in this case.

Although venlafaxine, a serotonin norepinephrine reuptake inhibitor (SNRI), can be used in pediatrics, it does not have as much evidence supporting its use in pediatrics, and according to the Texas Children's Medication Algorithm Project, it should be reserved for pediatric patients that fail two trials of SSRIs or can not tolerate SSRIs as a class.

3. Provide a follow-up schedule for pediatric patients initiating antidepressant therapy.

The previous FDA black-box warning regarding the risk of suicidality with antidepressants use in pediatrics recommended the following follow-up schedule for all pediatric patients initiating antidepressant therapy:

- ❑ Weekly face-to-face contact with the treating provider for the first 4 weeks
- ❑ Biweekly face-to-face contact with the treating provider at weeks 5 to 8
- ❑ Face-to-face contact with the treating provider at week 12
- ❑ After 13 weeks, the patient should be seen by the treating provider as clinically indicated.

The current FDA black-box warning no longer provides recommendations for the frequency of monitoring but instead states that treating providers should closely observe patients who are on antidepressant therapy. The best follow-up schedule can be debated, but pediatric patients should be seen at the start of therapy at the minimum of every 2 to 4 weeks to determine efficacy and to review adverse reactions of therapy until dose is optimized with minimal or tolerable adverse reactions. Depression is a disease state where studies have shown up to 50% of subjects demonstrated suboptimal adherence (missing greater than 30% of doses); therefore, close follow-up is often needed to ensure continued adherence to therapy. Once a patient is on an effective dose of an antidepressant and is in remission of symptoms, follow-up visits may be scheduled out longer, but some guidelines still recommend close follow-up with monthly visits in order to monitor for relapse of symptoms. As always, schedules should be individualized based on patient need and response. For this patient, the best follow-up schedule would be every 2 weeks until dose is optimized and she is in remission of symptoms, then monthly thereafter to monitor adherence and for relapse of symptoms.

4. Discuss nonpharmacologic therapy and patient education for pediatric patients receiving antidepressants.

Treatment of depression in pediatric patients should always include education and family involvement. This should include education about the causes, symptoms, and treatments of depression. Patients can also be referred for psychotherapy or talk therapy with a counselor. A Cochrane review of the literature found limited evidence that antidepressant medication was more effective than psychotherapy on obtaining remission from depression. There is also limited information that combination therapy is more effective than antidepressant medication alone. However, the conclusion was

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that there are limited data with conflicting results, which makes it difficult to determine the effectiveness of specific interventions. The American Academy of Child and Adolescent Psychiatry (AACAP) recommends the addition of psychotherapy when pharmacotherapy with antidepressants alone is ineffective. The addition of psychotherapy to pharmacotherapy for treatment-resistant depression has been found to be effective in a recently published trial. The most important issue to consider in designing a patient's treatment plan is patient and family preferences.

Pediatric patients initiating therapy with antidepressants should be provided with the following education:

- ❑ Review the importance of adherence to therapy for optimal effectiveness.
- ❑ Discuss with patient that it may take 4 to 6 weeks to see beneficial effects of therapy.
- ❑ Review the adverse reaction profile of antidepressants (e.g., headaches, GI adverse reactions, insomnia, agitation, irritability, appetite changes, and sexual dysfunction).
- ❑ Discuss risk and symptoms of serotonin syndrome (e.g., fever, hyperthermia, confusion).
- ❑ Explain the risks associated with abrupt discontinuation (e.g., flu-like symptoms, headache, insomnia) and the need to taper therapy before stopping.

The increased risk of suicidal thinking and behavior, as seen in patients under age 25 on antidepressants, needs to be reviewed. Patients and families should understand the possible increase in suicidal symptoms and thoughts, especially early in treatment and should understand the signs and symptoms. Patients and families should have a care plan in place in case the patient presents with the symptoms. They should also have available numbers for the suicide hotline and the local emergency crisis service. The patient is at greatest risk during the first 2 to 3 months of therapy initiation and during dosage adjustments; therefore, she is still at risk and the family needs to be aware.

5. Discuss a treatment plan and goals for pediatric patients with depression.

The treatment goal should be remission of symptoms and not just response to therapy. The patient is on the optimal dose of antidepressant when depressive symptoms have resolved and medication adverse reactions are absent or tolerable to the patient. While antidepressants are being titrated to the most effective dose, the patient is in the acute treatment phase. Once the patient's symptoms have resolved, the patient is in the continuation phase of therapy. Treatment should be continued for 6 to 12 months after resolution of symptoms. At that point it is determined if treatment should be continued as maintenance beyond 1 year. Patients who take a longer time to reach remission, those with two episodes of depression, or those with a severe episode will likely need therapy for longer than 12 months to prevent reoccurrence. This should be discussed with patients at the beginning of treatment so that they have an understanding of the duration of therapy needed.

6. Identify and provide a solution to the medication-related problem.

The patient is experiencing increased nervousness and anxiety secondary to the recent increase in the dose of fluoxetine. The change in therapy discussed above should alleviate this adverse reaction.

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CASE 8.4
Cerebral Palsy | Level 3

Mary Worthington

1. Identify subjective and objective findings indicative of spasticity or other complications associated with cerebral palsy.

SUBJECTIVE FINDINGS: The patient's chief complaint of pain, as defined by his mother, is a frequent complication of spasticity. Discomfort with movement, stiffness, and decreased mobility are symptomatic of spasticity. The patient's mother also reported a history of generalized spasticity with an early diagnosis of cerebral palsy. Spasticity is the most common motor abnormality associated with *cerebral palsy*, a term used to describe a range of developmental disorders of movement and posture. By definition, cerebral palsy must be ascribed to an injury or other disturbance in the developing fetal or infant brain. The majority of cases are acquired prenatally in contrast to this patient whose cerebral palsy is etiologically related to meningitis that occurred at 3 weeks of age. The noted intellectual disability, drooling, and constipation are consistent with additional common complications of cerebral palsy.

OBJECTIVE FINDINGS: The patient's pain has been evaluated using the **F**ace **L**egs **A**ctivity **C**ry and **C**onsolability (FLACC) behavioral pain scale. To assess a patient using the FLACC scale, the clinician observes the patient's behaviors in each of the five categories. Each category is scored from 0 to 2 using a rubric. A total score is then calculated between 0 and 10, with 10 indicating the highest assessment of pain. The patient's spasticity is generalized as it is found in all four extremities. The Modified Ashworth Scale rating of 3 indicates severe spasticity with difficult passive movement due to excessive increase in muscle tone. The inability to ambulate as noted by the rating of 5 in the Gross Motor Function Classification System (GMFCS) is also consistent with severe disease. The presence of contractures indicates prolonged spasticity. Truncal hypotonia and increased deep tendon reflexes are signs of abnormal neurologic control. Cognitive impairment is indicated by the developmental history findings of communication consisting of babbling and use of a communication board for five to six simple words. Excessive drooling with pooled saliva in the mouth are signs of impaired oral-motor function. The poor dentition is most likely related to excessive drool. The patient's gastroesophageal reflux disease (GERD) may contribute to the poor dentition. Impaired oral-motor function is a factor in the patient's failure to thrive. The patient's height and weight are at the 5th percentile on growth charts, and he requires enteral nutritional support. The osteopenia seen on x-ray is a signal for low bone mineral

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density that is related to the patient's nonambulatory condition and nutritional deficits. The decreased bowel sounds and palpable stool found on abdominal exam and the gas-diluted loops of bowel seen on x-ray are signs of constipation.

2. Evaluate the suitability of intrathecal baclofen therapy to treat the patient's increased spasticity.

It is important to prioritize addressing the patient's chief complaint of pain. A pain scale, such as the FLACC behavioral assessment tool, can be particularly helpful in the evaluation of level of pain in nonverbal patients. A revised version of the FLACC scale is available that includes behaviors associated with pain in children with cognitive impairment in the scoring rubric. Using the FLACC behavioral scale, this patient has been assessed as experiencing significant pain and discomfort. A thorough evaluation should be undertaken to identify the source of pain and to treat the underlying cause. Symptomatic pharmacologic treatment should also be provided. Because the patient is already experiencing problems with sedation and constipation, treatment with acetaminophen or a nonsteroidal anti-inflammatory drug would initially be preferred over an opioid. Although the patient has had only minimal relief with ibuprofen, intravenous ketorolac is a reasonable initial choice for symptomatic relief with careful monitoring for gastrointestinal and renal adverse reactions.

Hip dislocation or subluxation and presence of fracture have been eliminated as possible pain sources in the patient. The increased spasticity should be addressed as a possible cause of the pain. In addition to relieving pain, the goals of optimizing therapy for spasticity include maximizing the patient's motor function, decreasing the contractures he is experiencing, and easing the caregiving needs for his family. Pharmacologic treatment options include a change in oral pharmacotherapy, botulinum toxin treatment, or intrathecal baclofen. The patient has already experienced sedation with oral therapy. Clonazepam was discontinued due to this adverse reaction, and his mother notes undesirable sedation with oral baclofen after a recent increase in dose. The antinociceptive effects of tizanidine could be beneficial in the patient, but its use may be limited by a high incidence of somnolence and uncertainty in optimal dosing. The patient's current slight elevation in liver function tests and positive family history for hepatic disease reduce the viability of treating with oral dantrolene. Botulinum toxin therapy is indicated for localized spasticity while the patient has generalized disease affecting all extremities and major joints. If a particular muscle group can be identified as a source for pain or the increased stiffness, botulinum toxin treatment in those muscles would be beneficial. Based on his poorly controlled spasticity that is affecting quality of life and the limitations of other treatment options, the patient is a candidate for intrathecal baclofen therapy. In addition, efficacy of intrathecal baclofen is best supported in patients with severe spasticity (GMFCS levels IV or V). Performed in a monitored setting, a bolus-screening dose of intrathecal baclofen (25 mcg) can be used to confirm if the patient will likely respond to the continuous infusion given through the implantable pump. The duration of effect of the bolus dose is typically 8 hours and using the Modified Ashworth or similar scale at 2-hour intervals provides a measurable way to assess the patient's response. If a positive effect is found, it would also be important to determine the parents' abilities to manage this therapy and willingness to follow up as recommended prior to initiating continuous infusion treatment through the implantable pump.

3. Develop a plan to appropriately counsel the patient's parents on intrathecal baclofen therapy.

Education of the patient's parents will be a key determinant for the success of intrathecal baclofen therapy. They should clearly understand the goals and expectations of treatment, the steps in initiating therapy (screening, preoperative assessment, surgery, postoperative care, and follow-up), adjustments needed to other medications, potential adverse reactions of baclofen and how to manage them. The parents will need in-depth training on the implantable pump and documentation of the

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device model, serial number, catheter type, and length. They should be taught the signs and symptoms of baclofen overdose and withdrawal, as well as infectious complications and how to respond if symptoms arise. Overdose symptoms include sedation, lethargy, excessive hypotonia, and respiratory depression. In contrast, withdrawal is associated with changes in mental status, exaggerated rebound spasticity, muscle rigidity, elevated temperature, blood pressure lability, and seizures. Information on how the device may interact with other technology should also be provided. The parents should be instructed to carry an emergency information card that provides key instructions related to the therapy and potential complications.

4. Assess the pharmacologic treatment of drooling in the patient and provide additional nonpharmacologic treatment strategies.

Although evidence for efficacy is limited to small trials, glycopyrrolate is often the preferred choice when pharmacologic therapy is initiated for drooling in patients with cerebral palsy. Prior to initiating therapy, the patient's degree of drooling could be assessed using a modified Teacher's Drooling Scale. Repeated assessment could then be used to determine the patient's response and to aid with dose titration. Initial dosing with the oral solution is consistent with the dose utilized in a small study, although the dose could be titrated to 0.1 mg/kg/dose (2 mg for the patient, maximum 3 mg in the study) given 3 times a day. If the parents are unable to obtain the commercially oral available solution due to cost, a trial utilizing oral tablets could be initiated. The tablets could be crushed and mixed with the thickened foods that the patient is able to take as part of his diet. An initial dose of 0.5 mg 3 times a day with titration up to 2 mg 3 times a day would be appropriate with the tablet formulation. Constipation is a common adverse reaction that may limit the use of this drug in the patient. If the patient is unable to tolerate glycopyrrolate or has inadequate response to treatment, the patient would be a potential candidate to receive botulinum toxin injections into the salivary gland. Transdermal scopolamine would be an additional alternative treatment, but it is also associated with constipation. The use of transdermal scopolamine is also limited in younger pediatric patients as it is only available as a 1.5 mg patch that delivers 1 mg of scopolamine over a 72-hour period. The patient's GERD treatment should be optimized as it may be an aggravating factor for the drooling. GERD may also be contributing to the patient's nutritional difficulties and placing him at risk for recurrent upper respiratory infections. The current choice of ranitidine treatment for GERD may still be preferred over proton pump inhibitor therapy in light of the osteopenia seen on his x-rays, but his dose should be reassessed. If the patient still requires treatment for GERD, the appropriate dosing is 5 to 10 mg/kg (NTE 600 mg/day) divided into bid dosing. The patient's current dose is slightly low at 4.5 mg/kg/day.

Nonpharmacologic treatment is extremely important in managing the patient's drooling. A pediatric dentist should evaluate the patient as caries, malocclusion, or gingivitis may contribute to drooling. The patient's posture in sitting position should be assessed; and, if his head falls forward, his wheelchair should be adjusted to provide a slight recline while sitting. An occupational therapy consult is indicated to provide training in oral awareness and oral motor skills. In addition to decreasing drooling, improving the patient's oral health will have many benefits including reducing the risk of periodontal disease, caries, and halitosis.

5. Provide recommendations to further assess the need for additional treatments for low bone mineral density.

The patient is receiving the recommended amounts of supplemental vitamin D, as cholecalciferol due to its potential higher biologic potency, and dietary intake of calcium (1,000 mg elemental calcium in his enteral nutrition) for prevention of osteoporosis. He is also receiving additional calcium supplementation. Despite these interventions, osteopenia has been noted on his x-rays. Evaluations of serum 25-hydroxyvitamin D (25-OH-D) level and urine calcium/osmolality ratio would be beneficial to assess if his supplementation is maintaining these

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values at recommended levels (70 to 100 nmol/L for 25-OH-D and less than 0.25 for calcium/osmolality ratio). Physical therapy should be consulted to ensure the patient is performing appropriate weight-bearing activities. The patient has already been evaluated appropriately for fractures, but with his high risk, repeat assessments should occur with any further pain episodes. If a fracture occurs, the patient's z-score should be obtained through a dual-energy x-ray absorptiometry scan. If this value is less than –2.0, the patient will be diagnosed with osteoporosis, and treatment with a bisphosphonate could be considered.

6. Identify medication error and provide strategies for prevention.

Baclofen is unavailable commercially as an oral liquid. At home, the patient was receiving a 4 mL dose of a 5 mg/mL compounded preparation, or 20 mg. On admission, the patient has been ordered 4 mL of a 10 mg/mL compounded preparation, or 40 mg. The patient was already experiencing sedation on the home dose and is at risk for further problems with the erroneous dose. Strategies to prevent this medication error include (1) improving the medication reconciliation process on admission; (2) ensuring dosage evaluation by the pharmacist based on age and weight; (3) accurately documenting inpatient and outpatient medication dosing in the medical record; (4) educating parents on dose and dosing measurement of oral medications; (5) educating health professionals on error risk with extemporaneous formulations; and (6) advocating for commercial availability of suitable dosage formulations for children and standardized extemporaneous formulation concentrations.

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CASE 8.5
Seizure Disorders | Level 3

Jose Valdes and Jose A. Rey

1. What subjective and objective evidence supports the diagnosis of absence epilepsy?

SUBJECTIVE FINDINGS: The patient "spaced out" for about a minute at a time, during which he was conscious but unresponsive. The mother noted that his facial muscles appeared to jerk along with his eyelids briefly. Just before his episodes occurred, she saw him take a large long swallow. These subjective signs are characteristic of absence seizures.

OBJECTIVE FINDINGS: Although an EEG is not generally recommended in a patient with simple febrile seizure who is neurologically healthy, the patient is presenting with some signs that go beyond those of simple febrile seizures. The bedside EEG revealed small, subtle spike and wave patterns at 3 Hz generalized and symmetrical. The EEG also captured the characteristic pattern of regular and symmetrical spike-and-slow-wave complexes. This EEG pattern is characteristic of absence seizures.

Febrile seizures are divided into two categories (simple or complex) and are differentiated by their length of duration, presence of focal features, and number of episodes in 24 hours. *Complex febrile seizures* tend to last more than 15 minutes, have focal features, and occur more than once in a 24-hour period, while *simple febrile seizures* last less than 15 minutes, do not have focal features, and do not recur in a 24-hour period. Although this patient has a history of febrile seizures, he presents with features that fit closely with that of an absence epilepsy. *Absence seizures*, which are categorized as *generalized seizures*, are also known as *petit mal seizures* and are mostly present in childhood between the ages of 4 to 10. Absence seizures can start and stop abruptly. Patients with absence seizures typically present with blank stares and/or myoclonic jerks of the facial muscles and automatisms (lip smacking, swallowing, and clumsiness of a task at hand).

It is important to differentiate different seizure types. Most seizure types are placed in two categories: *partial* and *generalized* seizures. What differentiates the two is the spread of electrical activity—*partial* being focused to a specific area in one hemisphere while *generalized* affects both hemispheres and has electrical activity that is more widespread. Partial seizures are differentiated between simple and complex, with the main difference being the presence or loss of consciousness, respectively. Generalized seizures are further differentiated into absence, atonic, tonic-clonic, myoclonic, and infantile spasms.

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Atonic seizures are also known as *drop attacks* and present as a brief loss of muscle tone where self-injury can occur. *Tonic-clonic seizures*, also known as *grand mal seizures*, typically result in a loss of consciousness and present in two phases: a *tonic* phase where there is extension of the trunk and limbs and a *clonic* phase where there is synchronous muscle jerking. *Myoclonic seizures* result in rapid, brief muscle contractions that can affect the extremities, head, or trunk of the body and can be isolated or repetitive. *Infantile spasms* are considered as an age-specific generalized epilepsy mostly secondary to another disease and presents as an extension followed by flexion of the head, trunk, and/or limbs. *Febrile seizures* occur in children between the ages of 6 months through 6 years and typically present with clonic, atonic, or tonic symptoms.

Both absence and febrile seizures have shown a genetic component. In 10% to 15% of patients with absence epilepsy, there is a history of earlier febrile seizures. Febrile seizures can show a 25% to 75% risk of development if the patients have had a family history. One study has shown that in a family with several epileptics and the two main phenotypes being childhood absence and febrile seizures, it was noted that there could be an inherited gene mutation on chromosome 5 responsible for a $GABA_A$ receptor and gamma2 subunit. Other chromosomes have been implicated (10, 13, 14, and 15); however, research is still warranted in this area. Although the patient's father appears to have some form of epilepsy, the form remains unknown, which may be a risk factor for this patient. The patient also received a dose of DTaP, which in a large cohort study showed that febrile seizures were significantly increased the day of vaccination and 8 to 14 days following the MMR vaccine.

2. Develop a nonpharmacologic and pharmacologic regimen for the treatment of the patient's epilepsy.

NONPHARMACOLOGIC

Diet

The ketogenic diet introduced in 1921 is a popular therapy for patients who have refractory seizures consisting of a high-fat (90%), low-protein (8%), and low-carbohydrate (2%) dietary restriction. Other less stringent diets have been studied such as the modified Atkins diet introduced in 2003, which allows for a higher percentage of protein (30%) and carbohydrates (6%) and less fat (64%) and the low glycemic index treatment (LGIT) introduced in 2005, which is also a high-fat diet (60%) with restrictions on carbohydrate (10%) and protein intake (30%) with a focus on the glycemic index of foods and how they raise blood glucose.

Studies estimate a complete cessation of seizure activity in approximately 50% of patients utilizing the ketogenic diet or the modified Atkins diet and an approximately 90% improvement in 35% of patients. The LGIT shares similar efficacy achieving approximately 50% reduction in seizure frequency.

Gastrointestinal (GI) disturbances are the most common early onset adverse reactions. Less frequent early and later onset adverse reactions are weight loss; hypertriglyceridemia; transient hyperuricemia; hypercholesterolemia; symptomatic hypoglycemia; hypoproteinemia; hypomagnesemia; repetitive hyponatremia; low concentrations of high-density lipoprotein; lipoid pneumonia due to aspiration; hepatitis; acute pancreatitis; persistent metabolic acidosis; and prolonged QT interval.

Adverse reactions and efficacy would require monitoring monthly for patients that are started on a diet and every 3 months thereafter. Generally upon initiation of the ketogenic diet, patients may require hospitalization for careful monitoring, while a diet such as the modified Atkins diet and LGIT may be started outside of the hospital. Vitamin supplementation is generally necessary as the patient is maintained on a diet that increases the risk of nutritional deficiency and should be monitored. Reports of thiamine, vitamin D, vitamin C, selenium, and calcium deficiency have been found in children. If diets are initiated, a multivitamin and additional supplementation with calcium and vitamin D would be recommended.

Recommendation

Although there are data to support the use of diets for preventing seizures, it is not indicated

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for the initial treatment of seizures. Diets may be considered when pharmacologic therapy has not controlled the patient's seizures or when additional control of seizures is needed. Although some patients have been able to eliminate medications and stay seizure free with diet alone, utilizing appropriate pharmacotherapy is always the first step.

Young age is not a contraindication to surgery; however, this patient would not be a candidate for surgery as he has not met criteria.

PHARMACOLOGIC TREATMENT OF FEBRILE SEIZURES

Clinical trials have shown that antipyretics such as acetaminophen, given on a schedule or on an as-needed basis, are not effective in preventing a febrile seizure from occurring or preventing the recurrence of febrile seizures. When a child presents with febrile seizures, antipyretics are used to control the fever, while antiepileptics are used to treat the seizure if it persists for longer than 5 minutes. Febrile seizures that last less than 5 minutes and do not recur within 24 hours are generally self-limiting and do not typically require treatment. Although this patient has a history of simple febrile seizures, his symptoms are better described as absence seizures. Thus, the patient's fever should be treated symptomatically. It is recommended to use an agent that would not cause further hepatic insult.

Ibuprofen 200 mg po q 6 hr as needed, not to exceed four doses a day, would be the drug of choice for the treatment of the fever as the patient has hepatic impairment, and products that may cause further hepatic dysfunction (such as acetaminophen) should be avoided. The recommended dose of 200 mg may be given as 10 mL of a 100 mg/5 mL suspension or as two chewable tablets at 100 mg each for this patient. While the patient requires ibuprofen, monitoring of vital signs and body temperature every 6 to 8 hours as well as for signs of bleeding and rash is necessary. Adverse reactions associated with ibuprofen include abdominal pain, cramps, decreased appetite, nausea, rash, fluid retention, thrombocytopenia, anemia, and GI bleeding.

PHARMACOLOGIC TREATMENT OF ABSENCE SEIZURES

Evidenced-based treatment guidelines from the 2006 International League Against Epilepsy (ILAE) recommend childhood absence epilepsy be managed with ethosuximide, valproic acid, and lamotrigine. Based on randomized controlled trials, these medications are effective in children as monotherapy. In 2010, the results of a double-blind superiority trial comparing ethosuximide, valproic acid, and lamotrigine found increased efficacy for ethosuximide and valproic acid. As such, these two agents are preferred for monotherapy in the treatment of absence seizures. The results of that study showed a 53% and 58% freedom of seizures without intolerable adverse reactions for ethosuximide and valproic acid (no significant difference) with lamotrigine at 29% (significantly different). This gave level A evidence to ethosuximide and valproic acid for initial monotherapy and level C evidence for lamotrigine.

First-Line Agents

Ethosuximide (Zarontin®) (labeled indication)

Initial dose of 300 mg (15 mg/kg/day) po, can be given in two divided doses; however, the maximum initial dose is 250 mg, which may be given with 125 mg/2.5 mL of solution or syrup. Dose may be titrated upward every 4 to 7 days to a maintenance dose of 300 to 800 mg (15 to 40 mg/kg/day) per day, given in two divided doses. The maximum dose is guided by serum concentrations.

MONITOR—Obtain a complete blood count, urinalysis, and serum drug concentrations (goal trough of 40 to 100 mcg/mL) every week until a therapeutic concentration has been reached, then every 3 to 6 months thereafter. Liver function tests should be assessed weekly until normalized, then monthly for 3 months, and then every 3 to 4 months thereafter. Efficacy and adverse reactions should be evaluated and monitored monthly until a therapeutic concentration has been reached, then every 3 to 4 months thereafter.

ADVERSE REACTIONS—Adverse reactions include ataxia, difficulty concentrating, dizzi-

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ness, drowsiness, headache, irritability, lethargy, rash, diarrhea, abdominal pain, weight loss, agranulocytosis, and pancytopenia.

Valproic Acid (Depakote®) (labeled indication)

Initially, 200 to 300 mg (10 to 15 mg/kg/day) administered in one to three divided doses, is prescribed. It is available in a 250 mg/5 mL solution or 250 mg capsule. The dose is titrated by 100 to 200 mg (5 to 10 mg/kg/day) weekly as tolerated. Little data exist regarding safety and efficacy at doses above 60 mg/kg/day. Children 3 months to 10 years of age clear valproic acid 50% faster than children greater than 10 years old and adults, making the use of extended-release products difficult. Immediate-release formulations are preferred.

MONITOR—Obtain a complete count, liver function tests, and serum ammonia every week until an efficacious dose is reached, then monthly for 3 months, then every 3 to 4 months thereafter. Serum drug concentrations (goal trough of 50 to 100 mcg/mL) can be measured every week until therapeutic goals have been reached, then every 3 to 4 months thereafter. Monitor for adverse reactions such as nausea, vomiting, and weakness as this could be signs of hepatotoxicity. Efficacy and adverse reactions should be evaluated and monitored monthly until a therapeutic concentration has been reached and then every 3 to 4 months thereafter.

ADVERSE REACTIONS—Adverse reactions include headache, somnolence, dizziness, insomnia, nervousness, alopecia, nausea, vomiting, diarrhea, abdominal pain, dyspepsia, anorexia, thrombocytopenia, tremor, weakness, weight gain, blood dyscrasias, infection, flu-like syndrome, hyperammonemia, and elevated liver function tests. Black-box warnings include hepatotoxicity, increased hepatotoxicity risk in mitochondrial disease, pancreatitis, and fetal risk of teratogenicity. Children less than 2 years old are at an increased risk of hepatotoxicity, especially during the first 6 months of treatment, with risk decreasing in children greater than 2 years old.

Alternative Agents

Lamotrigine (Lamictal®) (off-label indication)

Initially, 10 mg (0.5 mg/kg/day) in two divided doses for 2 weeks is administered, followed by 20 mg (1 mg/kg/day) for 2 weeks. The dose is further titrated up by 20 mg (1 mg/kg/day) every 5 days until an effective dose is reached (maximum of 12 mg/kg/day).

MONITOR—Obtain a complete blood count every 2 weeks until an effective dose is reached, then every 4 to 6 months thereafter. Due to the risk of hepatotoxicity and given this patient's history, liver function tests should be assessed weekly until resolved, then monthly for 3 months, and then every 3 to 4 months thereafter. Efficacy and adverse reactions should be evaluated and monitored monthly until efficacy has been established and then every 3 to 4 months thereafter.

ADVERSE REACTIONS—The most common adverse reaction is nausea; other reactions are insomnia, somnolence, fatigue, impaired coordination, dizziness, rash, diplopia, agranulocytosis, and dyspepsia.

3. Assess which of the pharmacologic regimens to treat absence epilepsy would be best given the patient's hepatic impairment.

Due to the patient's impaired hepatic function, avoid further hepatotoxic medications until liver function is normalized. Unfortunately, the anticonvulsants with the best data to treat absence seizures are all hepatically metabolized to some extent. Clinically apparent hepatotoxicity from ethosuximide is rare and is generally part of a hypersensitivity reaction accompanied by fever, rash, facial edema, eosinophilia, or lymphadenopathy. Latency is typically 2 to 8 weeks, and although the product labeling warns of hepatic dysfunction, ethosuximide

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has not been linked to cases of chronic hepatitis or acute liver failure. Liver injury associated with ethosuximide resolves rapidly with discontinuation. Given its efficacy and low risk of hepatic complication, ethosuximide is recommended as first-line treatment for this patient. If significant hepatic impairment occurs during therapy, discontinuation should be considered. After titrating to the maximum tolerated dose, consider using lamotrigine as an adjunctive agent if seizures are not uncontrolled.

Lamotrigine is extensively and hepatically metabolized, and hepatotoxicity is estimated to occur in 1:2000 to 1:10,000 treated patients. Liver injury is generally part of an immune reaction similar to ethosuximide; however, it has resulted in a pattern of liver enzyme elevations usually ranging in severity from mild-to-moderate ALT elevations to a severe hepatitis and acute liver failure. Latency has been reported after ~26 days of therapy. Once hepatotoxicity is suspected or observed, stopping lamotrigine is recommended, and recovery is expected within 1 to 2 weeks. In the setting of hepatic impairment for the treatment of absence seizures, lamotrigine can be considered as adjunctive therapy. It does not require alternative dosing for hepatic impairment and would be a good alternative choice to use in the setting of mild hepatic impairment.

FEBRILE SEIZURE

Although the patient is on diazepam and use is intermittent, it is a benzodiazepine that is hepatically metabolized. The reduction of agents that are hepatically metabolized is preferred. A preferred strategy to preventing a febrile seizure is limited to an antipyretic as no other benzodiazepine is appropriate to administer. With regard to antipyretic use, it should only be utilized to reduce the fever, and a nonhepatically metabolized antipyretic should be utilized, such as ibuprofen.

4. Describe the goal of pharmacotherapy and the desired outcomes in treating the patient's seizure disorder.

Outcomes should be assessed frequently depending on the patient, type of seizures, and control of episodes. Therapeutic outcomes should consist of a decrease in the frequency of seizures with the end goal of eliminating seizures completely. Pharmacotherapy should be optimized as to improve the patient's quality of life and be free of adverse reactions. Withdrawal of anticonvulsant therapy may be considered if the patient has been seizure free for an average of 2 to 5 years, has a single type of partial/primary generalized tonic-clonic seizure, has a normal EEG with treatment, has a normal IQ, and has a neurological examination with nonsignificant findings. The decision to withdraw anticonvulsant therapy may be made with the physician, caregiver(s), and patient. Therapy should be tapered off over time—never abruptly stopped unless the patient is experiencing severe adverse reactions. Due to ethical limitations, very few studies examine the complications of untreated epilepsy. Some evidence points to an accelerating disease process with each seizure, meaning the onset of a seizure may beget future seizures, as in the case with generalized tonic-clonic seizures. For other types of epilepsy syndromes such as febrile and absence seizures, most patients will typically achieve remission of their seizures as they enter adulthood.

5. What information would you provide to the caregivers and child to help manage the symptoms?

Education is paramount to the patient or caregivers. Providing education can empower patients to take steps and manage the symptoms of their syndrome. Patients may be weary of beginning pharmacotherapy upon learning the different adverse reactions that the medications may have. It must be emphasized that it is a goal to reduce or eliminate the patients' seizures to increase their quality of life and to prevent adverse reactions as a result of drug therapy. If a patient is having intolerable adverse reactions, the physician should be contacted immediately to work with the patient/caregivers to find a suitable alternative treatment for the patient that may be better tolerated. Generally, most patients with a diagnosis of new onset epilepsy will become seizure free with the first or second medication administered. With medication, approximately 80% of patients are well controlled with either no or

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few seizures. Advise patients to follow up with a neurologist to ensure optimal care. Patients should be advised to avoid triggers that may cause a seizure such as stress, anxiety, lack of sleep, heavy alcohol intake, flickering lights (e.g., strobe lights, videogames), and illnesses that may cause a fever. Certain medications may reduce the seizure threshold; therefore, patients and caregivers should be advised to discuss the initiation of any new medications with a healthcare provider. Keeping a logbook of medications given, diet, activities prior to a seizure, duration of the seizure, and observations during the seizure (movement of extremities, facial jerking, etc.) may help to identify patient-specific triggers.

6. Describe how to best manage the medication-related problem.

The medication-related problem was due to the acetaminophen being given at a greater dose than recommended for a 4-year-old child weighing 20.8 kg. The patient received 400 mg per dose every 4 hours or 20 mg/kg/dose, which is above the maximum recommended dose of 15 mg/kg. The maximum dose is 75 mg/kg/day or 1,560 mg of acetaminophen per day. The patient was receiving approximately 100 mg/kg/day or 2,000 mg. To avoid further hepatic insult and treat future fevers, ibuprofen should be administered when necessary. Liver function tests should be monitored every 1 to 2 weeks outpatient to ensure a steady normalization of values.

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CASE 9.1
Acute Otitis Media | Level 1

Dominic Chan and Jennifer Le

1. What is the subjective and objective evidence that supports the diagnosis of AOM?

The diagnosis of AOM is based on clinical presentation with otoscopic examination. Middle ear effusion with acute onset of symptoms—including subjective symptoms of irritability (fussiness) and ear pulling suggestive of otalgia (or ear pain), and objective findings on otoscopy and of fever—are presenting attributes of AOM. On otoscopic examination, the tympanic membrane appears erythematous, cloudy, and bulging (which signifies presence of middle ear effusion). A bulging tympanic membrane is most characteristic of acute inflammation and, therefore, AOM. In contrast to AOM, otitis media with effusion (OME) is characterized by fluid in the middle ear without any signs or symptoms of infection and does not necessitate the use of antibiotic therapy.

2. What are the risk factors associated with AOM that are present in this patient?

Infants and young children are at increased risk for developing AOM because of the decreased angle of the opening of the eustachian tube that allows nasopharyngeal bacteria to ascend to the middle ear cavity and prevents adequate drainage of middle ear fluid. This anatomic feature of the eustachian tubes, along with other risk factors, is associated with the development of otitis media. Other risk factors for AOM evident in this patient:

- young age of less than 2 years
- recent upper respiratory tract infections (particularly caused by rhinovirus and adenovirus) promote bacteria replication and increase inflammation in the nasopharynx and eustachian tube, which subsequently facilitates bacterial entry into the middle ear space
- exposure to second-hand tobacco smoke
- pacifier use
- bottle-feeding
- daycare attendance

If modifiable, exposure to these risk factors should be minimized.

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3. Identify and resolve the medication-related problem.

The ibuprofen dose is low for this patient ~2.4 mg/kg per dose (common dose 5 to 10 mg/kg every 6 to 8 hours). This low dose may have contributed to the parent's claim of "slight" improvement with ibuprofen use at home. An alternative antipyretic medication should be considered (see next question).

The child may be experiencing increased bowel movements from ibuprofen use, although this can also be seen temporally with AOM. Discontinuation of ibuprofen and a change of therapy to acetaminophen 15 mg/kg (143 mg or 4.47 mL or round up to 4.5 mL) every 6 hours as needed for pain (maximum 75 mg/kg/day = 715 mg) may be warranted to help resolve the diarrhea. Notably, many liquid analgesics contain sorbitol as a sweetening agent, which may contribute to the undesirable effect of osmotic diarrhea (i.e., pulling water into the intestines). An over-the-counter antidiarrheal agent (e.g., loperamide) is *not* recommended for infants less than 1 year of age. For infants with diarrhea, maintaining adequate hydration with appropriate fluids (such as electrolyte-containing drinks but *not* juice or soda) and feeding are important. In addition, monitoring for a decrease in daily bowel movements to ensure resolution of this symptom and prevent complications is important.

4. Explain why this patient qualifies for initial observation or immediate pharmacologic treatment. If pharmacologic treatment is warranted, devise a plan for managing AOM.

Guidelines from 2004 recommended initial observation (i.e., without antibiotic therapy) in mild cases of AOM. Guidelines published in 2013 now recommend prescription of antibiotics *or* observation with close follow-up based on joint decision-making with the parent/caregiver for mild cases of AOM. If observation is selected, a mechanism must be in place to ensure follow-up and initiation of antibiotics if the child fails to improve within 48 to 72 hours. Antibiotic treatment should be initiated immediately in children with severe disease (i.e., toxic appearing, bulging tympanic membrane with apparent pus, persistent otalgia for greater than 48 hours, or temperature of 39°C or greater), or bilateral AOM. Specifically in this case, the patient is experiencing severe AOM evidenced by bulging tympanic membrane with pus, fever, and bilateral disease. Even if those symptoms were attenuated, the presence of bilateral AOM in a child younger than 2 years warrants treatment given the possible sequelae of untreated disease such as hearing loss and mastoiditis in a developing infant.

Antibiotic therapy is generally empiric and usually involves the use of oral agents with activity for the three most common organisms: *S. pneumoniae*, nontypeable *H. influenzae*, and *M. catarrhalis*. The patient has not been exposed to amoxicillin in the past 30 days, does not have purulent conjunctivitis, and does not have a penicillin allergy. As such, oral high-dose amoxicillin 80 to 90 mg/kg/day administered twice daily (i.e., 400 mg oral suspension twice daily, which can be provided as a suspension of 400 mg/5 mL) is the first-line treatment. Compared with twice-daily dosing, thrice-daily dosing of high-dose amoxicillin is associated with a significantly higher incidence of diarrhea. The standard duration of antibiotic therapy is 10 days (except if ceftriaxone or azithromycin is prescribed), particularly for children less than 2 years of age or those with severe disease. Clinical improvement, including fever and irritability, should occur within 48 to 72 hours after starting treatment (although symptoms may worsen during the initial 24 hours). If improvement is not evident within 48 to 72 hours, an evaluation for viral disease or a change in antibiotic therapy may be warranted.

Adjunctive therapy should address fever and otalgia. Acetaminophen, dosed as mentioned above, is effective for mild-to-moderate pain. Ibuprofen 5 to 10 mg/kg every 6 to 8 hours is *not* indicated in this patient due to the medication-related problem. Both acetaminophen and ibuprofen also serve as antipyretics. Pain should resolve within 48 to 72 hours of initiating treatment.

The two vaccines that have shown effectiveness in preventing AOM are the pneumococcal and influenza vaccines. The influenza

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vaccine may reduce the occurrence of AOM by 50%, particularly in young children attending daycare. The 7-valent pneumococcal conjugate vaccine, introduced in the United States in 2000, provides only a modest decrease in the occurrence of AOM compared with the flu vaccine. The 13-valent pneumococcal conjugate is now recommended for routine immunization in infants, and its effects on the incidence and epidemiology of AOM will remain uncertain until its use becomes widespread. Immunizations are up to date in this patient. It is important to verify whether children aged 6 months to 8 years received two doses of the influenza vaccine, separated by at least 28 days of each other.

5. What educational points should you provide to the anxious parents in optimizing the care of the patient's current and potential future infections?

Rapid and accurate diagnosis of AOM is recommended to ensure prompt and judicious antibiotic use. As such, the parents should be educated on the differences between AOM and OME. AOM is typically caused by bacteria, treatable by antibiotics (but for only certain diagnosis or severe disease), whereas OME is usually caused by viruses and hence making antibiotics ineffective. Furthermore, the parents should promptly report signs and symptoms of AOM to their child's healthcare provider to facilitate a rapid diagnosis. For the current infection, the presenting symptoms (e.g., fever, irritability) usually resolve within 2 to 3 days after initiation of antibiotics. However, middle ear effusion can persist for weeks to months in up to 70% of children.

Other educational recommendations include the need to finish the entire course of 10-day antibiotic therapy, smoking cessation or secondary smoke avoidance for the father (hand and face washing prior to contact with child after smoking), optimizing analgesic doses (acetaminophen 10 to 15 mg/kg administered orally every 4 to 6 hours, not to exceed five doses in 24 hours), avoiding ibuprofen until diarrhea resolves, and encouraging the parents to stay up to date with the vaccination schedule recommended by the Centers for Disease Control and Prevention and yearly seasonal influenza vaccines. The parents were "anxious" about the patient's fevers. A fever is defined as an elevation in body temperature exceeding 100°F by mouth (100.4°F rectally) and is part of the natural human body response to infection. Fevers do not harm the patient and can be managed using appropriate doses of acetaminophen. Lastly, the parents may believe that warmed olive oil may be used to sooth the child's otalgia—it is important to explain the source of the pain is beyond the tympanic membrane, where the olive oil cannot (and should not) pass.

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CASE 9.2
Skin and Skin Structure Infections | Level 1

Jennifer Le

1. What is the subjective and objective evidence that supports the diagnosis of cellulitis and requirement for hospitalization?

The diagnosis of cellulitis is based on the following clinical findings:

SUBJECTIVE FINDINGS: Increased leg erythema and swelling, fever per mother, and pain

OBJECTIVE FINDINGS: Fever (T_{max} 39.5°C); slight hypotension (BP 105/68 mm Hg), leukocytosis (WBC $16 \times 10^3/\mu L$); elevated neutrophils (PMN 79.1%); left lower leg positive for erythema (nondemarcated), warmth, tenderness, induration, and edema (usually without suppuration or necrosis); and x-rays of femur, tibia, and fibula negative for soft tissue and bone involvement

The abrasion that the patient experienced 2 weeks ago may have introduced a bacterial inoculum to trigger the current cellulitis of his lower left leg. This patient presents with moderate-to-severe cellulitis that is accompanied by fever and leukocytosis with elevated neutrophils, which necessitate hospitalization for appropriate treatment.

2. Explain why the patient has cellulitis rather than erysipelas and necrotizing fasciitis by differentiating these types of skin and skin structure infections.

In contrast to cellulitis that affects the deep skin layers including the dermis and cutaneous tissue, *erysipelas* involves the epidermis and dermis. Erysipelas clinically manifests as intense erythema with sharply demarcated, palpable borders that may progress to bullae formation with severe necrosis.

Necrotizing fasciitis is a potentially life-threatening infection that affects the subcutaneous tissues. Although only mild swelling and erythema may be observed on the infected site, patients commonly report disproportionate pain. Systemic symptoms (e.g., oral or tympanic temperature of ≥38°C or hypothermia, tachycardia, hypotension, confusion, leukocytosis) are common with necrotizing fasciitis, and patients may appear critically ill.

This patient has a nondemarcated skin infection marked by warmth, tenderness, and edema, and it is accompanied by systemic symptoms. There is no evidence of soft tissue involvement from the radiographic tests or report of disproportionate pain (scale 3/10). As such, the diagnosis of cellulitis supports this patient's clinical presentation.

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3. What are the most likely pathogens causing cellulitis in this patient?

Most cases of cellulitis are caused by pathogenic bacterial skin colonizers, including *S. aureus* (methicillin-susceptible) and group A *Streptococcus* (also known as *S. pyogenes).* In cases of cellulitis complicated by abscess formation, community-associated methicillin-resistant *S. aureus* (CA-MRSA) should be strongly suspected. This patient has a serious cellulitis with notable fever, hypotension, and leukocytosis; hence, the pathogens suspected to cause infection are *S. aureus* (including CA-MRSA) and group A *Streptococcus,* which may have been introduced by the skin abrasion that the patient experienced 2 weeks ago.

4. Design an empiric antibiotic treatment regimen (including drug name, dose, frequency, and duration) that is appropriate for this patient.

Given the serious presentation of cellulitis with systemic symptoms (i.e., fever, slight hypotension, and leukocytosis), the patient should be hospitalized to receive parenteral antibiotic that adequately covers CA-MRSA at least empirically until culture and susceptibility data become available. The primary therapeutic goal is to cease the spread of, or reduce the redness, edema, and pain caused by the cellulitis within 48 to 72 hours after antibiotic initiation. Vancomycin 60 mg/kg/day IV (or 340 mg IV every 6 hours) is recommended empirically and should be adjusted based on therapeutic drug monitoring. If bone involvement, bacteremia, or other serious infections are definitively excluded, vancomycin dosing should be adjusted to meet an appropriate pharmacodynamic target to successfully treat cellulitis. If the patient is clinically stable, clindamycin 40 mg/kg/day (or 300 mg IV every 8 hours) is an alternative but should be used only if the local MRSA resistance rate is <10%. Clindamycin is a good option for transition to oral therapy for susceptible strains. Linezolid 30 mg/kg/day (or 230 mg every 8 hours) should be reserved for severe infections (e.g., septic shock) or when alternative antibiotics are contraindicated. Antibiotic therapy should be tailored toward culture and susceptibility data. The antibiotic treatment duration for cellulitis cases requiring hospitalization is typically 10 to 14 days, depending on the patient's response. Improvement (or cessation of worsening) of clinical symptoms (e.g., fever, erythema) and laboratory abnormalities (i.e., WBC and PMNs) should be observed within 48 to 72 hours after antibiotic initiation. Transition to oral antibiotic therapy should occur when systemic symptoms resolve and significant clinical improvement at the site of the cellulitis is observed. In addition to antibiotic therapy, leg elevation and warm compresses 3 times daily will help the healing process. Topical antibiotic therapy is not indicated in this patient.

5. Identify, resolve, and provide parent education for the medication-related problem.

Based on gender and age, the patient is within the 5th percentile for weight and 80th percentile for height. The patient is experiencing loss of appetite and significant weight loss (~10 pounds) from the use of Adderall XR, which is a combination of the stimulants amphetamine and dextroamphetamine. The Adderall XR dose was increased 2 weeks ago to maximum dose of 30 mg per day. To minimize these significant adverse reactions, the Adderall XR dose may be decreased to the minimum effective dose as low as 10 mg per day (if possible). Notably, a heart murmur was reported on physical exam. Without any previous history of murmur, an echocardiogram should be performed to confirm a true murmur. All medications for ADHD possess a risk for cardiac effects, with the highest risk from stimulants. However, the stimulants are most effective for ADHD so a true murmur should be ruled out prior to considering other medications to treat ADHD.

The parents should be instructed to monitor and help improve the patient's appetite, diet, and weight. The patient should follow up with a healthcare professional to ensure achievement of weight gain, improvement of appetite, and for an echocardiogram to confirm a true heart murmur. If not improved, Adderall XR may need to be discontinued and another medication (e.g., clonidine or atomoxetine) may be initiated to manage his ADHD.

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CASE 9.3
Parasitic Infections | Level 2

Heather L. Girand

1. What subjective and objective evidence supports the diagnoses of pinworm infestation and head lice in this patient?

Pinworm and head lice infestation are distinctly different parasitic infections that both occur commonly in children of all socioeconomic classes. Pinworm, or *Enterobius vermicularis*, is the most common worm infection in the United States. The prevalence is as high as 50% in school-aged children along with their families or caregivers and in institutionalized children. Infection occurs only in humans and is caused by ingestion of pinworm eggs that subsequently hatch and mature in the small intestine. The mature female worm migrates to the colon and lays eggs around the anus usually at nighttime. Infection is easily spread to others via contamination of hands, clothing, bedding, and toilet seats with eggs.

Head lice infection, or *Pediculus humanus capitus*, is another common infection in school-aged children and their families or caregivers with up to 12 million infestations each year in the United States. It spreads through head-to-head contact with an infested person, wearing clothing worn by an infested person, using infested brushes or towels, or lying on bedding or pillows used by an infested person. Infection is propagated through adult lice that crawl from person to person. The adult females lay eggs (nits) that attach to hair shafts near the scalp and hatch within 1 week. Nymph forms then mature into adult lice about 7 days after hatching. The adult lice require frequent blood feedings from the scalp several times each day and will die within 1 to 2 days without a host. Risk factors for both pinworm infestation and head lice include young age because of the lack of attention to proper hygiene and spread of infection through close contact.

SUBJECTIVE FINDINGS OF PINWORM INFESTATION: The patient presented with a chief complaint of perianal itching and burning that began recently. The itching and burning were significant enough that they interfered with her sleep last night. She attends elementary school and daycare, and she recently spent prolonged time in close proximity with young family members, which are both risk factors for pinworm infestation.

OBJECTIVE FINDINGS OF PINWORM INFESTATION: On physical exam, she has perianal and perivaginal erythema and skin excoriations near her anus. The erythema extends to her groin, which indicates a possible secondary bacterial infection. The "tape test" is positive, indicating that pinworm eggs were present. This test is performed

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by touching the perianal area with transparent tape in order to collect eggs that can be viewed under a microscope.

SUBJECTIVE FINDINGS OF HEAD LICE: The patient had no subjective findings related to head lice. As noted previously, her age, school and daycare attendance, and recent camping trip where she shared sleeping quarters with young family members are risk factors for contracting head lice.

OBJECTIVE FINDINGS OF HEAD LICE: The office nurse observed that the patient has been scratching behind her ears, and nits have been noticed at the base of her hair shafts behind the left ear and in the occipital area. These are common areas to find head lice. The presence of nits alone that are within ¼ inch of the hair shaft base is also suggestive of head lice infestation, but the diagnosis can only be confirmed if live lice are observed. This patient has been exhibiting symptoms (itching), but many children are asymptomatic.

2. Assess the use of hydrocortisone for this patient's symptoms related to pinworm infestation.

Hydrocortisone is a commonly used topical corticosteroid available without a prescription (0.5% and 1%) to treat inflammation and itching associated with various conditions including atopic dermatitis, contact dermatitis, insect bites, and psoriasis. Its action, in theory, is mediated by immune suppression of cytokines that promote vasodilation, inflammation, and pruritus. It should not be applied to broken or infected skin because systemic absorption is enhanced when the skin barrier is disrupted, and it can mask symptoms of local infections, potentially causing them to worsen if they are not treated with appropriate anti-infective therapy. This patient has perianal excoriations caused by scratching, and she has evidence of cellulitis that has spread to the groin area. Use of topical hydrocortisone should be discouraged because its vasoconstrictive and anti-inflammatory properties can make monitoring for improvement of the secondary bacterial infection more difficult. Treatment of the underlying condition with anthelminthic therapy will relieve the perianal pruritus.

3. Develop a pharmacologic treatment plan for this patient's pinworm infestation and head lice.

The goals of treatment for pinworm infestation are to relieve symptoms, eradicate the infestation, and prevent reinfection and transmission to others. Anthelminthic therapy is recommended and is available with or without a prescription. Pyrantel pamoate is a nonprescription oral anti-infective that causes neuromuscular depolarization and paralysis of adult worms, which subsequently detach from the intestinal wall and pass in the stool before their eggs can be laid on the perianal skin. One initial dose is given but is often followed by a repeat dose in 2 weeks because the medication only kills worms and not eggs or developing larvae. The second dose will kill worms that hatched from eggs still present after the first treatment dose; a second dose should be given if it is recommended by a primary care provider upon repeat examination, particularly if perianal itching or pain continue. Albendazole is an anthelminthic agent that interferes with the worm's ability to store glycogen and produce other energy sources, leading to its eventual death. It can be used in patients who fail pyrantel therapy or whose healthcare providers prefer its use; however, it requires a prescription, is more expensive, requires administration with high-fat meals, and may have more adverse reactions than pyrantel pamoate. It also usually requires two doses given 2 weeks apart to reduce recurrence. Mebendazole is similar to albendazole but is no longer available in the United States.

This patient should receive 187 mg (11 mg/kg) of pyrantel base per dose in an amount that is easily measurable; the dose should be rounded up to 250 mg given as 5 mL of the 50 mg/mL suspension. Pyrantel pamoate can be taken with or without food at any time of the day. Adverse reactions are uncommon but, if present, are usually mild and short-lived; they can include nausea, vomiting, diarrhea, abdominal cramps, and tenesmus. If she is still experiencing symptoms of perianal itching or pain 2 weeks after

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her initial treatment, she should be reexamined and retreated if pinworms are still present.

The goal of treatment for head lice is to eliminate the infestation by killing the adult lice and nymphs typically in conjunction with manual nit removal from hair shafts. Pediculicides are generally recommended for most children and are available with or without a prescription. In geographical areas with low rates of resistance to nonprescription agents, either 1% permethrin or pyrethrins are first-line therapies. Permethrin cream rinse has some advantages over pyrethrins, such as less allergenicity and the presence of a long-acting residue that can make retreatment unnecessary. Proper use of these products in conjunction with nit removal increases their success, but treatment failure may occur if resistance is present. Prescription agents such as malathion, benzyl alcohol, spinosad, and ivermectin are recommended for children who have failed nonprescription therapy or who reside in communities where resistance to nonprescription agents is high.

In this patient, live lice were not observed but nits were found at the hair shaft base and she was symptomatic, so pharmacologic therapy is recommended. She can be treated with permethrin 1% rinse, and her mother should be taught how to properly use this medication. She should wash her daughter's hair with a nonconditioned shampoo, rinse with water, and towel dry the hair so that it is damp but not wet. After shaking the permethrin bottle well and protecting her daughter's eyes with a washcloth or towel, she should apply the product so that it completely saturates the hair and scalp, ensuring that it is applied behind the ears and on the back of the neck. After 10 minutes, she should rinse the hair with warm water, towel it dry, and comb out the tangles. Adverse reactions to permethrin are usually mild and may include tingling, burning, itching, or redness at the site of application.

The mother should also remove the nits with a nit comb that is provided with the permethrin product. Although this is a tedious process that can take an hour or longer depending on how much hair the child has, it is highly effective for preventing persistent infestation. The combing should be done while the hair is still damp. The hair should be parted into four sections and combed out one section at a time. In each section of hair, a 1-to-2 inch wide hair strand should be combed out beginning as close to the scalp as possible. After combing each strand and removing the nits from the comb with a tissue, the strand should be pinned back away from the noncombed hair; this process should be repeated until each section has been completely combed out. The tissue that was used to remove the nits from the comb should be sealed in a plastic bag and discarded. The child's head should be inspected daily for nits and, if present, they should be removed with the nit comb. If live lice are found between 7 and 10 days after the initial permethrin application, a second treatment can be given.

4. Provide pertinent education for the parents regarding management of pinworm infestation and head lice.

A successful response to pinworm treatment is heavily dependent on processes used to prevent reinfection. Hygienic measures such as changing and washing bed linens and undergarments in hot water daily, keeping fingernails clean and cut short, discouraging nail biting and scratching, and frequent hand washing for all family members are critical for preventing self-inoculation and spread to close contacts. Daily vacuuming of carpeted areas and wet mopping of other surfaces are recommended for removing eggs from the home environment. Bathing or, preferably, showering in the morning is recommended for infected individuals in order to reduce the egg burden on the skin. Family members with evidence of infection or whose household members experience repeated symptomatic infection should also be treated with anthelminthic therapy. Bed-sharing should be avoided until infestation is eradicated.

In contrast to the easy spread of pinworm infection, head lice are mainly transmitted through direct head-to-head contact or sharing of items such as hats, brushes, towels, or pillows. Reinfection is uncommon because head lice cannot survive long after they fall from the scalp, but environmental measures are still recommended to prevent spread. All clothing and washable

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items used by the infected person should be washed in hot water and dried on high heat, if possible, in order to kill lice and nits; items that cannot be laundered should be dry cleaned or sealed in plastic bags for 2 weeks. Carpets and furniture where the infested individual has recently been can be vacuumed, but the risk of transmitting lice in this manner is low; insecticidal sprays are not recommended. In addition, all household members and close contacts of the infested individual should be checked and treated if active infestation is present. Prophylactic pharmacologic treatment is recommended for those who have recently shared (within 2 days) or currently share bed quarters with the infested individual.

5. Identify a medication-related problem in this patient and provide a recommendation for its management.

Inappropriate antibiotic prescription for cephalexin as patient is allergic to cefprozil is the medication-related problem.

This patient developed hives while taking cefprozil, so other cephalosporins should be avoided unless there are no other appropriate treatment options. Nonpurulent cellulitis is usually caused by methicillin-susceptible staphylococci or group A streptococci, and many antibiotics can be used to target these bacteria. Common empiric oral agents include cephalexin, dicloxacillin, clindamycin, and amoxicillin/clavulanate. Cross-reactivity between penicillins and cephalosporins is less common than historically reported because of improvements in medication purity and changes in beta-lactam side chains that frequently induced allergy. Cross-reactivity between penicillins and first-generation cephalosporins is now approximately 5% or less as compared to 2% or less with second-, third-, or fourth-generation cephalosporins. Cross-reactivity to other beta-lactams in cephalosporin-allergic patients ranges from 8% to 50% and may be as low as 1% or less when challenged with oral agents as compared to parenteral routes. Knowledge regarding beta-lactam side chains can be helpful when selecting potential therapies in patients with beta-lactam allergies: cross-reactivity is more likely with agents that have similar or identical side chains. This patient developed hives while taking cefprozil; its side chain is identical or similar to cephalexin, cefadroxil, and amoxicillin. Alternative agents that could be prescribed to treat this patient's cellulitis include dicloxacillin or clindamycin in order to avoid beta-lactams with similar chemical structures to cefprozil.

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CASE 9.4
Pneumonia | Level 2

Dominic Chan and Jennifer Le

1. What is the subjective and objective evidence that supports the diagnosis of community-acquired pneumonia?

SUBJECTIVE FINDINGS: The most common symptoms associated with pneumonia in children are fever and cough, which is present in this patient.

OBJECTIVE FINDINGS: Vital sign abnormalities include a fever, pulse oximetry of 89% on room air, and an elevated WBC count with high neutrophil count. On physical exam, the patient demonstrates nasal flaring and dyspnea. The diagnosis of pneumonia is supported by this clinical presentation along with the presence of abnormal chest radiography that is consistent with pneumonia with effusion versus empyema.

Signs of respiratory distress in children with pneumonia include tachypnea, dyspnea, retractions, grunting, nasal flaring, apnea, altered mental status, and pulse oximetry measuring less than 90% on room air. The patient has some of these signs of respiratory distress, warranting hospitalization for management of her pneumonia.

The patient's pneumonia is classified as community-acquired, as opposed to healthcare-associated pneumonia, due to the absence of recent exposure to healthcare facilities and antibiotic use in the past 90 days.

One possible unifying diagnosis for this patient is a viral illness causing her acute gastroenteritis prior to current hospital admission with subsequent development of a superimposed lobar pneumonia. Although uncommon, children with pneumonia may have nonspecific symptoms of emesis and abdominal pain.

2. What are the most likely pathogens causing community-acquired pneumonia in this patient, and what complication associated with pneumonia is presented?

Both viruses and bacteria can cause pneumonia. Given the age of the patient and lobar nature of the pneumonia, bacterial pneumonia is more likely than viral pneumonia. The potential bacterial pathogens causing pneumonia in this patient are the following:

- *Streptococcus pneumoniae*
- *Staphylococcus aureus*, including methicillin-resistant (MRSA), especially if empyema present

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- Group A *Streptococcus*, particularly with possible recent enteric viral infection
- Atypical pneumonia, primarily *Mycoplasma pneumonia, Chlamydophila pneumoniae*

Pneumonia may progress to pulmonary complications that consist of pleural effusion, empyema (or collection of pus), abscess, and necrotizing pneumonia. The chest x-ray of this patient reveals left pleural effusion with opacity that is consistent with pleural effusions or empyemas. Admission to the intensive care unit and/or mechanical ventilation may be required if any of these complications occur. Children infected with typical bacterial organisms, such as *Streptococcus pneumoniae*, and those co-infected with viruses are more likely to develop pleural effusions. Pathogenic bacteria most commonly associated with empyema are *S. pneumoniae*, *S. aureus* (including MRSA), and group A *Streptococcus.* Determination of a definitive organism is typically acquired via direct sampling of the infected space through various means (e.g., thoracentesis, assistance by interventional radiology).

3. Design a pharmacologic treatment regimen and monitoring plan for the treatment of complicated community-acquired pneumonia in this patient.

Pneumonia is the greatest cause of death in children worldwide; it is important to treat this disease to reduce the mortality and morbidity associated with it. Complications of untreated pneumonia include the development of lung abscesses, bacteremia, infections of other organ systems, respiratory failure, and death.

Children presenting with respiratory distress due to suspected complicated pneumonia (as is evident in this patient) should be hospitalized for appropriate management of infection. For presumed bacterial pneumonia in a fully-immunized patient with a potential life-threatening complication such as an empyema, empiric therapy should provide coverage for *S. pneumoniae* and *S. aureus,* including possible MRSA. Hence, the antibiotic regimen should consist of a third-generation cephalosporin such as ceftriaxone 100 mg/kg/day with a maximum dose of 2 g/day (1 g IV q 12 hr) with vancomycin 15 mg/kg (375 mg) IV every 6 hours (which is equivalent to 60 mg/kg/day) or clindamycin 40 mg/kg/day (250 mg) IV every 6 hours. Clindamycin should *not* be used empirically when community resistance rates for MRSA exceeds 10%. The criteria to select an antibiotic with activity versus atypical organisms for a hospitalized patient is not well defined and depends on the clinical presentation as well as the patient's age. It is suggested that patients greater than 7 years old or patients receiving outpatient treatment benefit the most from macrolide or tetracycline antibiotics.

A thoracentesis or video-assisted thoracoscopic surgical procedure may be necessary for drainage of the empyema to promote lung function. If performed, bacterial cultures should be obtained during the surgical intervention to tailor definitive antibiotic therapy. Literature extrapolated primarily from adult data suggests vancomycin concentrations should be measured and doses adjusted to achieve the pharmacodynamic target of AUC/MIC ≥400; currently, most institutions utilize trough concentrations, so trough levels of 15 to 20 mcg/mL serve as an adequate surrogate for this target. Ceftriaxone can be used as monotherapy for treatment of empyema caused by susceptible *S. aureus,* and *S. pneumoniae* (even when the penicillin MIC is ≥4 mcg/mL). If MRSA is isolated and therapy with clindamycin is considered, a double-disk diffusion test (also called a *D-test*, in which one disk is impregnated with erythromycin and another disk with clindamycin) should be performed to exclude the presence of inducible clindamycin resistance mediated by the *erm* gene. The duration of therapy is typically 10 to 14 days, although longer duration of up to 4 weeks may be necessary in children who have pneumonia complicated with empyema.

Daily assessment of clinical improvement (increased level of activity, increased appetite, decreased fever, consistent pulse oximetry measurements of greater than 90% on room air, normalization of laboratory measurements such as WBC count and markers of inflammation as applicable) should be observed, with a goal of improvement within 48 to 72 hours after initiation of empiric antibiotics. If this goal is not

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met, further investigation of alternative reasons for respiratory dysfunction is warranted.

The patient should also be monitored daily for adverse drug reactions associated with vancomycin use, such as red man syndrome (limited if infusion time extended), renal dysfunction via serum creatinine and ototoxicity; clindamycin, including diarrhea (in particular caused by *C. difficile*); or ceftriaxone, such as gallbladder sludging and hepatic abnormalities via hepatic function tests including bilirubin. As with any beta-lactam, CBC should also be monitored for irregularities.

4. Identify and resolve the medication-related problem.

The most likely cause for her microcytic anemia is an iron deficiency; however, because she is having dark stools, a contributing factor may be blood loss. As such, this patient's anemia is possibly secondary to a gastrointestinal bleed from the use of ibuprofen. The discontinuation of ibuprofen and initiation of a parental proton-pump inhibitor (PPI), such as pantoprazole 1 mg/kg (25 mg) IV daily, are warranted given the decreased hemoglobin and dark tarry stools suggestive of an upper gastrointestinal bleed. Intravenous PPI therapy is typically continued for at least 72 hours after the bleeding has stopped before transitioning to oral therapy. The use of a PPI in conjunction with a third-generation cephalosporin may predispose the patient to develop *Clostridium difficile* colitis, thus monitoring for loose stool is warranted.

Daily CBC should be monitored. Duration of therapy depends on the resolution of the upper gastrointestinal bleed or an alternative diagnosis for the anemia. Treatment for the prevention of rebleeding is unlikely necessary, given the transient limited nonsteroidal anti-inflammatory drug (NSAID) use.

Parents should be educated to observe for any signs and symptoms of rebleeding, including bloody vomit or mucus, bright red or dark tarry stools, and/or persistent abdominal pain. The parents should also avoid the use of NSAIDs in their child for the relief of pain or fever. Instead, the parents may use acetaminophen-containing products.

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CASE 9.5
Kawasaki Disease | Level 3

Jennifer Le

1. What subjective and objective evidence supports the diagnosis of Kawasaki disease?

The current diagnostic criteria for Kawasaki disease consist of unexplained fever persisting for 5 days or more, with four or more primary clinical features that are present in this patient as subjective and objective findings. Although laboratory abnormalities are not required for a diagnosis of typical Kawasaki disease, some are highly suggestive of disease and are listed below.

SUBJECTIVE FINDINGS: A 23-month-old Chinese girl presented with the following:

- 7-day history of persistent fever
- 4-day history dry lips and red tongue
- 4-day history of nonbloody diarrhea (which is worsening)
- 1-day history maculopapular rash developing on her chest and abdomen

OBJECTIVE FINDINGS: On physical exam, patient has bilateral, nonexudative conjunctivitis, edema of the hands and feet, and diffused blanching macular rash over abdomen. Abnormal laboratory findings consistent with Kawasaki disease are leukocytosis (WBC count 26.7 × 10^3/μL), normocytic anemia (Hgb 10.1 g/dL, Hct 29.7%), thrombocytosis (platelets 877 × 10^3/μL), elevated CRP and ESR (20.2 mg/dL and 56 mm/hr, respectively), sterile pyuria (WBC count 16/hpf with negative bacteria on Gram stain), hypoalbuminemia (albumin 2.5 g/dL), and elevated serum aminotransaminases (AST/ALT 56/79 units/L). Furthermore, diagnostic tests to confirm Kawasaki disease, including chest x-ray, influenza, and respiratory syncytial virus, are negative.

Another possible etiology for the patient's clinical presentation, particularly worsening diarrhea, is gastroenteritis that can be caused by a virus or bacteria (including *Shigella*, *Campylobacter*, *E. coli*, and *Yersinia*). However, gastroenteritis is unlikely due to the marked CRP elevation. The diarrhea is also unlikely caused by *C. difficile* because the patient was not recently hospitalized or exposed to broad-spectrum antibiotics (excluding amoxicillin). Other infections, including pneumonia and pyelonephritis, may elevate WBC count; however, diagnostic work-up consisting of normal chest x-ray and urinalysis suggests otherwise.

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2. What risk factors evident in this patient support the diagnosis of Kawasaki disease?

Ethnicity and young age are common risk factors for Kawasaki disease. The incidence is highest among children of Asian or Pacific Islander descent. In addition, up to 90% of cases occur in patients between 6 months and 5 years of age.

3. What additional diagnostic test would you recommend to rule out the common complications of Kawasaki disease?

Although a self-limiting disease, Kawasaki disease can progress to cardiac complications including coronary artery aneurysms, congestive heart failure, myocarditis, pericarditis, and arrhythmias. Coronary artery aneurysms, the major cardiac complication, occur in one-fourth of untreated children with Kawasaki disease versus 4% with therapy, emphasizing the importance of adequate treatment. Prompt initiation of appropriate therapy, particularly within 10 days of fever onset, may prevent cardiac morbidity progression and mortality. To ascertain the degree of cardiac involvement, an echocardiogram should be performed within 2 weeks after fever onset and repeated 6 to 8 weeks later to confirm therapeutic response.

4. Recommend and explain the benefit(s) of pharmacologic treatment during hospitalization and at discharge. Provide the therapeutic goals and monitoring plans for this patient.

Based on guidelines developed by the American Heart Association and the American Academy of Pediatrics, the standard therapy for Kawasaki disease is a single dose of intravenous immune globulin (IVIG) and high-dose aspirin initiated as soon as the diagnosis is made or within the first 10 days of illness. The benefits of IVIG are its anti-inflammatory properties to support resolution of fever and prevention of coronary artery aneurysm. Although it does not decrease the development of coronary abnormalities, aspirin is used for its additive anti-inflammatory and antiplatelet activities.

A single, high-dose infusion of IVIG at 2 g/kg (22 g infused over 8 to 12 hours for this patient), compared with low doses over 4 to 5 days, has been shown to produce a greater decrease in the duration of fever and length of hospital stay as well as faster resolution of laboratory findings for acute inflammation. In addition, for its anti-inflammatory and antiplatelet activities, high-dose aspirin 80 to 100 mg/kg/day administered orally (243 mg in this patient, which equates to 3 of 81 mg chewable tablets every 6 hours, not to exceed 4 g/day) is recommended until 48 to 72 hours after fever resolves. Alternatively, high-dose aspirin can be continued until 14 days after illness onset, with at least 48 to 72 hours after fever cessation. This is followed by low-dose aspirin 3 to 5 mg/kg/day (i.e., ~40 mg [½ of 81 mg chewable tablet] orally once daily) for its antiplatelet effect and is continued until resolution of ESR or platelet count or until 6 to 8 weeks after illness onset to rule out cardiac abnormalities. Aspirin, including the low-dose therapy, should be discontinued in patients with influenza or varicella infection to avoid Reye syndrome. Clopidogrel can be temporarily substituted for aspirin in this situation.

Additional medications to prevent thrombosis formation may be necessary if coronary involvement is present. At this time, none of the following medications are indicated in this patient. They should be considered based on the result of the echocardiogram and clinical progress. The use of antiplatelet, anticoagulation, or combination therapies will depend on the degree of coronary involvement. For mild, asymptomatic cases, low-dose aspirin is recommended. The addition of other antiplatelet drugs (e.g., clopidogrel or dipyridamole) to low-dose aspirin to further enhance activity may be more effective in moderate cases with evidence of enlarged coronary artery aneurysm. Anticoagulation using warfarin (with initial unfractionated heparin as bridge therapy), in combination with low-dose aspirin, is indicated when the risk of thrombosis is high, particularly in rapidly enlarging coronary aneurysms.

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Adding methylprednisolone to standard therapy should be considered in cases at high risk of cardiac involvement. Ibuprofen or naproxen may be used for several weeks to treat prolonged arthritis or arthralgia, if present. However, their use should be avoided during low-dose aspirin therapy because they may interfere with the antiplatelet action of aspirin.

Alleviation of inflammation in the coronary artery wall and prevention of coronary thrombosis are the primary therapeutic goals for this patient. Other targeted therapeutic outcomes are to achieve defervescence, reverse presenting symptoms, normalize any irregular laboratory tests, and prevent mortality from myocardial infarction or arrhythmias. Hospitalization is required for diagnostic evaluation and IVIG administration. To ascertain the degree of cardiac involvement, an echocardiogram should be performed within 2 weeks after fever onset and repeated 6 to 8 weeks later to confirm therapeutic response. Referral to pediatric cardiology is required for all patients with Kawasaki disease, particularly after hospital discharge for close monitoring. Follow-up echocardiograms every 1 to 2 years after diagnosis may be warranted to determine the status (e.g., improving or worsening) of coronary aneurysms and to evaluate for other cardiac conditions.

5. Identify, resolve, and provide parent education for two medication-related problems.

Amoxicillin-induced allergic rash perceived by parents—The development of the maculopapular rash on the patient's chest and abdomen is most likely triggered by her Kawasaki disease as this illness is characterized by the presence of prolonged fever, pleomorphic rash, and a constellation of other symptoms and laboratory abnormalities suggestive of inflammation. The patient does not have an allergy to amoxicillin, and proper education should be provided to the parents (i.e., not claim the patient is allergic to amoxicillin) to avert preclusion of beta-lactams for future use. In exploring allergic reactions further, a true IgE-mediated allergic reaction is incongruous with the patient experiencing the rash 2 days after the initiation of amoxicillin therapy (i.e., reaction usually occurs within 1 hour of the first dose administration), particularly after previous exposure when sensitization developed.

Diarrhea caused by amoxicillin is improbable because the diarrheal episodes initiated 1 day prior to the use of amoxicillin.

First-generation antihistamine (diphenhydramine) paradoxical excitation in young children—Many parents are unaware that first-generation antihistamines are *not* indicated for insomnia in children <12 years old and that manufacturers do not promote their systemic use for any indication in children <6 years old. Diphenhydramine can cause a paradoxical excitement in children and, in rare cases, adults. The FDA specifically advises against its use to sedate children. Furthermore, although diphenhydramine is effective in relieving symptoms of rhinitis and sneezing associated with the common cold, FDA discourages its use in children <2 years of age due to serious safety concerns. Both parents should be educated on the safe and effective use of diphenhydramine and other over-the-counter cough and cold medications.

6. What educational points should you provide to the parents regarding the safe use of intravenous immune globulin, aspirin, and future vaccinations?

Overall, IVIG is generally well tolerated, but infusion-related reactions (consisting of flushing, hypotension, nausea, vomiting, fever, chills, pruritus, malaise, myalgia, and chest tightness) may occur during administration. These reactions generally occur 30 minutes to 1 hour after initiation of the infusion and usually resolve when the infusion is slowed or temporarily discontinued. Pretreatment with acetaminophen and diphenhydramine may help abate these infusion-related reactions. In addition, antibodies contained in IVIG may interfere with the immune response to certain live virus vaccines (e.g., varicella). As such, live vaccines should be deferred for at least 11 months in patients receiving IVIG treatment. Repeat immunization is necessary if the live virus vaccine was administered within 14 days before, or at the same time of IVIG

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administration. Other routine childhood vaccinations (e.g., pneumococcal conjugate vaccine) should be administered, without any change, as recommended by the Centers for Disease Control and Prevention.

The parent should administer aspirin with food to minimize stomach upset. The patient may be at increased risk of developing adverse reactions from aspirin use, consisting of elevation of transaminases, transient hearing loss, and Reye syndrome. Children taking long-term aspirin therapy should receive their annual influenza vaccine, specifically the inactivated form. Household contacts should also receive their annual influenza vaccine using either the live or inactivated formulations. In addition, aspirin, including the low-dose therapy, should be discontinued in patients with influenza or varicella infection to avoid Reye syndrome. Furthermore, aspirin should be avoided in the 6 weeks after varicella vaccination. Clopidogrel 1 mg/kg/day (maximum 75 mg) can be temporarily substituted for aspirin in these situations. Over-the-counter ibuprofen and other nonsteroidal anti-inflammatory drugs should be avoided in combination with low-dose aspirin because of their ability to interfere with the antiplatelet cardioprotective action of aspirin.

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CASE 9.6
Human Immunodeficiency Virus Infection | Level 3

Heather L. Girand

1. What subjective and objective evidence supports the diagnosis of HIV in this child?

SUBJECTIVE FINDINGS: The patient presented with a chief complaint of fever, cough, difficulty breathing, and poor feeding. These symptoms are consistent with an acute pulmonary infection and could be indicative of *Pneumocystis jiroveci* pneumonia, given this patient's clinical condition. Infants with HIV infection are commonly diagnosed with *P. jiroveci* pneumonia. Developmental delay is frequent in untreated HIV in infants; this child is not crawling or babbling, according to his foster mother. The infant's biological mother engaged in risky behaviors, had a known history of sexually transmitted infections, and did not undergo prenatal care; therefore, maternal-to-child transmission of HIV is of great concern in this case.

OBJECTIVE FINDINGS: Findings consistent with *P. jiroveci* pneumonia are fever, tachypnea, cough, respiratory distress (presence of nasal flaring and retractions), and hypoxia. Lung auscultation revealed bilateral basilar rales and chest radiography obtained at the urgent care center showed bilateral diffuse parenchymal infiltrates, which are consistent with *P. jiroveci* pneumonia. Presence of *P. jiroveci* pneumonia or other opportunistic infections is a strong indicator of HIV infection or severe immune compromise; this child has leukopenia and a CD4 count <15%. Other findings consistent with HIV infection in this child include failure to thrive (weight-for-age <2%, length-for-age ~2%), microcephaly (head circumference <2%), malnutrition (hypoalbuminemia), candidal infections (thrush and diaper dermatitis), and anemia. Laboratory evidence of HIV is demonstrated by a positive HIV antibody test, positive Western blot, and HIV RNA level of 190,000 copies/mL.

2. Assess the treatment plan for this patient's pneumonia and recommend changes to the regimen, if necessary.

Definitive diagnosis of *P. jiroveci* pneumonia can be made only by isolating the organism from pulmonary tissue or fluid, which usually requires an invasive procedure such as bronchoscopy with bronchoalveolar lavage (BAL). Treatment can begin prior to definitive testing because results are often positive for at least 72 hours after initiating antimicrobial therapy. Intravenous trimethoprim/sulfamethoxazole (TMP-SMX) is the drug of choice and is dosed as 15 to 20 mg/kg/day (of trimethoprim component) divided every 6 hours. As the pneumo-

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nitis resolves, children with mild-to-moderate disease who do not have malabsorption or significant diarrhea can be converted to oral TMP-SMX (same daily dose) for a total course of therapy of 21 days. Intravenous TMP-SMX has been difficult to procure in recent years because of manufacturing problems. Although it is currently available in specific vial sizes, future availability may be sporadic and hospitals should proactively identify alternative agents for use when it is not available. For this child, the initially prescribed antibiotic regimen is appropriate for his weight and renal function (37.5 mg IV every 6 hours is equal to 20 mg/kg/day). The diagnosis should be confirmed through appropriate microbiologic testing, and the child should be monitored for improvement (normalization of respiratory rate, oxygen saturation, breath sounds, temperature, and blood gasses; disappearance of nasal flaring and retractions) and, if possible, converted to oral therapy to complete a 21-day course. Oral TMP/SMX can be used when there is evidence of clinical improvement and the child is able to tolerate oral therapy (no malabsorption or significant vomiting or diarrhea). If he is unable to tolerate TMP/SMX or does not respond to it after 5 to 7 days of therapy, then IV pentamidine (4 mg/kg once daily) can be given.

This child is at risk for *P. jiroveci* pneumonia but could also be infected with other organisms such as *Streptococcus pneumoniae*, *Haemophilus influenzae* (nontypable or type b), *Mycoplasma pneumoniae*, or viral pathogens. His lack of immunization against common pathogens in community-acquired pneumonia (CAP) and his immunocompromised state increase the risk for pneumonia caused by these organisms. Empiric treatment for CAP (IV ampicillin 200 mg/kg/day divided every 6 hours with or without IV azithromycin 10 mg/kg/day) should be initiated concomitantly with TMP/SMX until the diagnosis of *P. jiroveci* pneumonia is confirmed because the rates of TMP/SMX resistance in *S. pneumoniae* and *H. influenzae* are high.

Corticosteroid therapy is recommended for children with moderate-to-severe *P. jiroveci* pneumonia because it has been demonstrated to reduce the rates of respiratory failure and mortality. Clear indications for corticosteroid treatment are PaO_2 <70 mm Hg or an alveolar-arterial gradient of >35 mm Hg on room air. This child was hypoxic on presentation to the urgent care center and placed on supplemental oxygen before transfer to the hospital. His venous blood gas revealed mild respiratory acidosis with some metabolic compensation, and there was concern for respiratory failure. Empiric initiation of corticosteroid therapy is appropriate for this child given the high likelihood of *P. jiroveci* pneumonia and the severity of presentation, but some healthcare providers may opt to delay steroid therapy if the diagnosis can be confirmed in the next 72 hours. If steroid therapy is initiated, prednisone or prednisolone can be used and are dosed as 7.5 mg (1 mg/kg/dose) for this child given twice daily for 5 days, followed by 3.75 mg (0.5 mg/kg/dose) given twice daily for 5 days and 3.75 mg (0.5 mg/kg) once daily through day 21. Prednisolone is preferred over prednisone in the liquid formulation because of its improved palatability. If oral therapy is not an option, IV methylprednisolone can be administered using 1 mg/kg dose every 6 hours on days 1 to 7, followed by 1 mg/kg/dose bid on days 8 to 9, then 0.5 mg/kg/dose bid on days 10 to 11, and 1 mg/kg/dose daily on days 12 to 16.

Finally, because of this child's immune status, secondary prophylaxis for *P. jiroveci* should be initiated after the acute infection has resolved. No antimicrobials used to treat *P. jiroveci* completely eliminate it from the body; therefore, prophylaxis is required after treatment for active infection in immunocompromised persons unless criteria to discontinue secondary prophylaxis are met. Prophylaxis with oral TMP-SMX (dosed as 5 mg/kg/day [of TMP] or 5 mg/kg 3 times weekly on consecutive days) should continue until the child is treated with antiretroviral therapy for at least 6 months and has CD4 ≥15% or CD4 ≥500 cells/mm^3. For this child, the dose should be 37.5 mg (TMP component) once daily. If he is unable to tolerate TMP/SMX, oral dapsone (2 mg/kg/day) can be used for secondary prophylaxis.

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3. If antiretroviral therapy will be initiated, what is an appropriate regimen for this child?

Guidelines recommend initiating combination antiretroviral therapy (cART) in all HIV-infected infants less than 12 months of age, regardless of viral load, CD4 percentage, or clinical status. The goals of therapy are to reduce morbidity and mortality, preserve or restore immune function, attain viral suppression, maintain optimal growth and development, minimize adverse reactions, prevent resistance, and provide the best possible quality of life. Young children are at high risk for rapid disease progression and death, and early treatment is preferred to achieve faster immune recovery and reduce morbidity. In order to achieve optimal therapeutic success, clinicians must assess and discuss adherence issues with the child's caregivers before beginning treatment. This can be performed while laboratory tests are pending, but decisions regarding initial therapy must occur early in order to provide the most optimal outcome for young children infected with HIV. This child should be treated with cART in order to suppress viral replication, try to restore immune function, reduce morbidity from opportunistic infections, and enhance growth and development as much as possible. Many clinicians would postpone initiation of cART until the acute pneumonia resolves and antiviral resistance testing can be performed.

Guidelines recommend cART with at least three active drugs from two different classes in order to minimize development of antiretroviral resistance. Proper drug selection is vital for treatment-naïve children because it can affect future treatment options if or when resistance and virologic failure occur. The recommended initial regimen for children less than 3 years of age includes two nucleoside reverse transcriptase inhibitors (NRTIs) plus ritonavir-boosted lopinavir. This regimen was shown to be superior for virologic suppression over two NRTIs plus nevirapine in young treatment-naïve children. Alternatively, nevirapine can be substituted for ritonavir-boosted lopinavir in children who cannot tolerate it (the liquid formulation has poor palatability) and were not previously exposed to nevirapine, including the use of maternal peripartum single-dose nevirapine. The preferred dual NRTI regimens in children include abacavir or zidovudine plus lamivudine or emtricitabine. The combination of abacavir plus lamivudine provides more potent virologic suppression and better growth rates than zidovudine plus lamivudine; in addition, in children where viral suppression is achieved after 24 weeks of therapy, abacavir can be dosed once daily instead of twice daily. The major drawback with abacavir is its association with life-threatening hypersensitivity reactions in patients with certain HLA genotypes: before initiating abacavir, genetic testing for HLA-B*5701 must be performed and, if positive, abacavir should not be used. In children, the combination of zidovudine plus lamivudine has the most extensive clinical data and is generally well tolerated. Major drawbacks to this regimen are bone marrow suppression (neutropenia and anemia) and lipodystrophy/metabolic changes caused by zidovudine. Emtricitabine can be substituted for lamivudine and has the advantage of once daily dosing, but there is less clinical experience with emtricitabine in young children.

Recent reports of "functional cure" or "remission" with early cART have generated interest about the role of early intense antiretroviral treatment and its durability on viral suppression. A child from Mississippi was treated with zidovudine, lamivudine, and high-dose nevirapine beginning at 30 hours of life before confirmation of HIV diagnosis. She was treated only until 18 months of age when her mother discontinued antiretroviral therapy for unknown reasons, but she has remained free from viremia for over 2 years. Similarly, a child from California was treated with the same cART regimen within 4 hours of birth and was found to be free from virus within 11 days. This patient has remained on cART and has had no evidence of viremia for almost 1 year. Both of these children were born to mothers who received no prenatal care and were treatment-naïve. Until the duration of response can be monitored in these patients and studied in other children, early intense antiretroviral therapy or interruption of cART after achieving viral response is not recommended.

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For this child, a regimen of ritonavir-boosted lopinavir 120 mg (16 mg/kg) bid, abacavir 60 mg (8 mg/kg) bid, and lamivudine 30 mg (4 mg/kg) bid is an appropriate first-line choice because he is treatment-naïve. He should be screened for the HLA-B*5701 allele before initiating abacavir; if positive, zidovudine could be substituted. Abacavir is preferred for this child because he is already anemic from HIV infection and zidovudine could exacerbate this condition. All of these medications are available in liquid formulations, are administered twice daily, and should be given with meals such as breakfast and dinner (ritonavir-boosted lopinavir oral solution is more bioavailable when given with food) in order to enhance adherence to therapy.

4. Provide a monitoring plan for this patient that takes into account current therapies and healthcare problems.

This patient should be monitored for improvement of pneumonia as demonstrated by oxygenation status, temperature, respiratory rate, and radiographic changes. Clinical improvement should be evident by days 4 to 5 of therapy. Initial worsening in the first 3 to 5 days of antimicrobial therapy may indicate an inflammatory reaction rather than clinical failure, so it is recommended to continue initial therapy for at least 4 days before changing therapy if clinical failure is suspected. Adverse reactions to TMP-SMX include rash, bone marrow suppression, gastrointestinal changes (nausea, diarrhea), and renal impairment. If an urticarial rash or signs of Stevens-Johnson syndrome occur, TMP-SMX should be discontinued and not reinitiated. Adverse reactions to corticosteroid therapy include hyperglycemia, behavioral changes (hyperactivity, insomnia, irritability), fluid retention, and increased blood pressure.

Children treated with cART should be monitored for efficacy, toxicity, and tolerability as well as medication adherence with biweekly to monthly clinic visits and laboratory evaluation during the first few months of therapy. This child had many laboratory values obtained on hospitalization, but other baseline values that are needed include hepatic transaminases and a lipid profile. Within the first 2 weeks of initiating cART therapy, it is recommended to evaluate children for medication adverse reactions and emphasize adherence because it is essential for minimizing viral resistance, particularly when therapy is first initiated. Palatability of liquid formulations can create problems with adherence in young children; administering doses with food, using flavoring agents, and implementing reward systems can help parents or caregivers of children who struggle with oral liquid preparations. After beginning cART therapy, the following parameters should be monitored within 2 to 8 weeks to assess initial treatment response: HIV viral load, CD4 percentage, serum chemistries, and complete blood cell count with differential. Lipid panels should be evaluated every 6 to 12 months. Goals of therapy are to achieve virologic suppression (defined as plasma HIV RNA below the level of quantification (<20 to 75 copies/mL depending on the assay) after 6 months of therapy) and immunologic response (as noted by achievement and maintenance of CD4 percentage (or CD4 count beginning at 5 years of age) that is above the age-specific range for severe immunodeficiency). A significant reduction in viral load should be evident by 4 to 8 weeks of therapy. It should be noted that some infants with high initial HIV RNA levels may respond more slowly to therapy with >200 copies/mL after 6 months, but with continued treatment and persistent adherence they can achieve virologic suppression, particularly with ritonavir-boosted lopinavir regimens. If this child is treated with abacavir, lamivudine, and ritonavir-boosted lopinavir, he should be monitored for these adverse reactions: dyslipidemia, hyperglycemia, fat maldistribution, elevated liver enzymes, gastrointestinal intolerance (vomiting, diarrhea), rash, fever, and pancreatitis.

It is important to assess virologic, immunologic, and clinical responses separately because treatment failure or progression of disease can be independent of virologic response, particularly in advanced stages. Virologic failure is defined as the inability to achieve viral suppression or viral rebound after suppression is documented. Immunologic failure is characterized by an incomplete response to cART (failure to increase CD4 percentage by 5% in those who

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present with CD4 <15%). Clinical failure is observed when children are unable to grow at a normal rate despite adequate nutrition, experience recurrent infections, or exhibit significant neurocognitive impairment. Decisions to alter cART are based on the responses in each category above.

Other healthcare problems that must be addressed in this patient are dehydration, failure to thrive, candidal infection, and lack of immunizations. At presentation to the urgent care center, the child was noted to be tachycardic with a recent history of poor oral intake, fever, and tachypnea, which all increase his risk for dehydration. He was given an appropriate fluid bolus and then transferred to the hospital where he continued to display signs of moderate dehydration (irritability, tachycardia, dry mucous membranes, mottled skin, capillary refill 3 seconds, elevated BUN and serum creatinine). Another fluid bolus should have been given to stabilize his circulation (normalize heart rate; reduce capillary refill to <2 seconds). Implementation of IV fluids was appropriate for his clinical condition, but the rate should be altered based on his current clinical condition. Given this child's fluid deficit and maintenance needs, he should receive 5% dextrose with ½ normal saline and 20 mEq KCl/L at an initial rate of 70 mL/hr for 8 hours followed by 50 mL/hr for the next 16 hours. After 24 hours, the IV fluid rate should be reduced to a maintenance rate (30 mL/hr) if he appears well hydrated and continues to require fluid therapy.

Children with HIV are at risk for growth delay and should be followed closely by a registered dietician experienced in managing patients with this condition. In addition, children with advanced disease commonly experience wasting and malnutrition that further compromise the immune system. This child is severely malnourished (weight-for-age <2%, length-for-age ~2%, hypoalbuminemia) and is not ingesting enough calories to support normal growth. His diet consists primarily of formula, which provides 48 kcal/kg/day, approximately half of the recommended daily caloric intake for an 11-month-old child. A nutrition consult should be obtained with the goal of providing an age-appropriate diet that is high in protein and calories to improve his nutritional status and meet the high-caloric requirements of patients with AIDS.

This patient is also experiencing candidal infection as a result of his immune suppression. He has oral thrush and a candidal diaper rash that should be treated with antifungals to hasten their resolution. The treatment of choice for uncomplicated thrush is oral nystatin suspension (2 to 4 mL applied directly to the tongue and then swallowed 4 times daily) for 7 to 14 days. Oral fluconazole 30 mg (4 mg/kg/day) daily for 7 to 14 days can be used if topical nystatin fails, but it is best to avoid azole antifungals in patients receiving protease inhibitors, when possible, because of the drug–drug interactions that occur. This child is not currently receiving antiretroviral therapy, but it should be initiated as soon as possible once the child is stable because of his significant immune suppression. Similarly, candidal diaper rash can be treated with topical nystatin ointment 4 times daily for 7 to 14 days.

Finally, this child is significantly delayed in his immunizations, which places him at risk for vaccine-preventable diseases. Most vaccines recommended for routine use in children can be administered safely to children with HIV infection, but their immunologic response may not be optimal. This child should receive the following inactivated vaccines that he is behind on, according to the Advisory Committee on Immunization Practices catch-up schedule (found at http://www.cdc.gov/vaccines/schedules/hcp/child-adolescent.html): hepatitis B, diphtheria/tetanus/acellular pertussis, inactivated poliovirus, pneumococcal conjugate, *Haemophilus influenzae* type b, and seasonal influenza. It is recommended to monitor hepatitis B antibody titers in HIV-infected children 1 to 2 months after completing the vaccine series to determine if a protective response was achieved or if a second vaccine series is required. In addition, he should receive the hepatitis A vaccine series beginning at 12 months of age. Finally, although live vaccines can be considered in certain patients, neither varicella vaccine nor measles/mumps/rubella vaccine should be administered to this child until the CD4 percentage is greater than or equal to 15% for at least 6 months.

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5. Discuss the medication-related problem in this patient and provide a recommendation for its management.

Inappropriate use of dextromethorphan in an infant is the medication-related problem.

Dextromethorphan is a commonly used cough suppressant that exerts its activity by raising the cough threshold. There is a lack of evidence to support its efficacy in children for acute cough caused by respiratory tract infections. In addition, reports of toxicity and death in infants and young children treated with nonprescription cough and cold medicines (including dextromethorphan) led the FDA's Nonprescription Drugs and Pediatric Advisory Committee and the FDA to recommend against their use in children under 6 years of age and under 2 years of age, respectively. Despite these 2007 recommendations and product labeling changes that warn consumers not to use over-the-counter cough and cold medications in children under 4 years of age, reports of infant fatalities continue to surface. Dextromethorphan toxicity may occur despite "normal" doses in patients with reduced metabolic capacity of dextromethorphan O- and N-demethylation that is dependent on cytochrome P450 2D6 activity: this can occur as a result of genetic polymorphisms or as a slow maturation process of this metabolic pathway in children. Given the lack of data to support its efficacy in children and the concerns for toxicity, dextromethorphan should not be used in young children and its use should be discontinued in this patient.

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CASE 9.7
Meningitis | Level 3

Heather L. Girand

1. What subjective and objective evidence supports the diagnosis of bacterial meningitis in this patient?

SUBJECTIVE FINDINGS: The patient presented with a chief complaint of fever (39.1°C) and irritability for the past 3 days. Early this morning, she vomited and became lethargic and is now difficult to arouse. Young children with meningitis often present with nonspecific symptoms, which can lead to misdiagnosis.

OBJECTIVE FINDINGS: On physical exam, she is febrile (39.7°C in ED), lethargic, and irritable during examination, particularly during the neck exam. The lumbar puncture results that are suggestive of bacterial meningitis are hazy color, low glucose (cerebrospinal fluid [CSF] to serum ratio ≤0.4), elevated protein, and leukocytosis with a predominance of neutrophils. This patient has evidence of a traumatic spinal tap (RBC >100,000/mm^3), which can cause xanthochromia and falsely elevate the protein and WBC count in the CSF sample. For every 1,000 RBCs in the CSF caused by a traumatic tap, there is an artificial increase of one WBC and 1 mg/dL of protein. After correcting for these values in this patient, the WBC and protein counts are still elevated and suspicious for bacterial meningitis. Other laboratory features that suggest a serious bacterial infection in this patient include hyperglycemia, metabolic acidosis, an elevated peripheral WBC count with a left shift, an elevated procalcitonin, and an elevated C-reactive protein.

Key diagnostic information used to elucidate the cause of meningitis is the Gram stain and culture of the CSF. Because this child's symptoms are consistent with meningitis, a CSF sample was obtained. The CSF Gram stain was negative, and the culture is pending. It should be noted that this patient received three doses of amoxicillin prior to the lumbar puncture obtained in the emergency department. Antibiotic pretreatment, whether oral or parenteral, can affect the positivity rate of blood and CSF cultures in children with bacterial meningitis, whereas Gram stain results do not appear to be significantly affected. However, Gram stain has a fairly low sensitivity rate (67%) for detecting bacterial meningitis and cannot be used alone to exclude infection. Other tests that may be performed to determine the cause of infection include latex agglutination and polymerase chain reaction, particularly in children who have been pretreated with antibiotics before a CSF sample was obtained or whose Gram stain and culture results are negative.

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2. Develop a treatment plan for this patient's meningitis.

It is important to distinguish between bacterial and viral meningitis in order to minimize morbidity and mortality while avoiding unnecessary antibiotic use. Once it is determined that bacterial meningitis is present or until it can be ruled out when a bacterial cause is suspected, empiric antibiotic therapy is required as soon as clinically possible. The empiric regimen that is selected should be based on patient age and other clinical characteristics, such as predisposing conditions, immunization status, medication allergies, and geographical antimicrobial susceptibilities. In children 1 to 23 months of age, the most common bacterial pathogens are *Streptococcus pneumoniae*, *Neisseria meningitidis*, *H. influenzae*, *Streptococcus agalactiae*, and *Escherichia coli*, where the latter two are less common after 2 months of age. Universal childhood immunizations introduced in developed countries have nearly eradicated *H. influenzae* type b (Hib) meningitis and dramatically reduced invasive disease caused by vaccine serotypes contained in the 7-valent pneumococcal conjugate vaccine (PCV-7). Non-PCV-7 serotypes are now the leading cause of bacterial meningitis in this age group; however, PCV-7 was replaced by the 13-valent pneumococcal conjugate vaccine (PCV-13) in 2010. Its use may impact the prevalence of pneumococcal meningitis, and its effect must be monitored through epidemiologic surveillance studies. In young children, meningococcal meningitis is most commonly caused by serogroup B for which no currently licensed vaccine is available in the United States but is approved for use in Europe, Australia, and Canada. Outbreaks of serogroup B meningococcal disease at the University of California, Santa Barbara, and Princeton University in 2013 and 2014 prompted the FDA to allow emergency use of the meningococcal B vaccine for at-risk students and staff.

Based on this child's age and lack of vaccinations, the most likely causative organisms are *S. pneumoniae* and *N. meningitidis*. Although she has not been vaccinated against Hib, near complete eradication of this pathogen in the United States and protection from herd immunity make this organism a less likely cause of her infection. In addition, she has a recent history of AOM, which can lead to meningitis, particularly when caused by pneumococci. Therefore, she should receive empiric antibiotic therapy that covers both pneumococci and meningococci. The Infectious Disease Society of America (IDSA) treatment guidelines recommend initiation of parenteral vancomycin plus either cefotaxime or ceftriaxone in order to provide adequate coverage for potentially drug-resistant pathogens. Ceftriaxone is contraindicated in neonates who are hyperbilirubinemic or who require calcium-containing intravenous (IV) fluids, and it should be used with caution in patients with concurrent hepatic dysfunction and significant renal disease or patients with presence of or risk factors for biliary stasis or sludging. The goals of treatment are to eradicate the infecting organism and to prevent or manage complications. This patient should be monitored for improvement (normalization of vital signs, return to baseline mental status and behavior, and normalization of laboratory values including WBC count, procalcitonin, and C-reactive protein [CRP]) and the development of complications such as seizures and hearing loss. She should also be monitored for medication adverse reactions including hypersensitivity and renal dysfunction. Repeat lumbar puncture is recommended if there is a lack of improvement or worsening after 48 hours of appropriate antibiotic therapy, in patients with pneumococcal meningitis caused by penicillin- or cephalosporin-resistant strains (particularly if dexamethasone was administered), and in neonates with meningitis caused by gram-negative bacilli.

For this patient, IV vancomycin 15 to 20 mg/kg/dose (150 mg) every 6 hours and IV ceftriaxone 50 mg/kg/dose (480 mg) every 12 hours can be initiated. If CSF culture and sensitivity results allow, antimicrobial therapy can be narrowed to treat the specifically identified pathogen. If vancomycin therapy will be continued for more than 48 hours, a target vancomycin trough of 15 to 20 mcg/mL is recommended. The duration of antibiotic therapy is dependent on the causative pathogen: a 7-day treatment course is recommended for meningococcal and *H. influ-*

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enzae meningitis while a 10- to 14-day course is recommended for pneumococcal meningitis.

Another issue to address that is related to meningitis is this patient's lack of immunization, which predisposes her to these serious infections. Healthcare providers must investigate why she has not been vaccinated and attempt to convince her parents that she should be immunized. If her parents agree to have her vaccinated, she should receive the following vaccines once she is no longer acutely ill: hepatitis B vaccine; diphtheria, tetanus, and pertussis vaccine; Hib vaccine; PCV-13; inactivated poliovirus vaccine; measles, mumps, and rubella vaccine; varicella vaccine; and hepatitis A vaccine. She is also a candidate for an annual influenza vaccine. The catch-up immunization schedule that is approved by the Advisory Committee on Immunization Practices should be consulted for recommendations on vaccine timing for the vaccines that require a dosing series (http://www.cdc.gov/vaccines/acip/index.html).

3. Assess the use of dexamethasone for this patient's meningitis.

Neurologic sequelae resulting from meningitis occur in up to 49% of children despite antibiotic treatment, ranging from behavioral disorders to seizures and loss of hearing or vision. The presence of bacteria and the host immune response lead to neuronal damage, inflammation, thrombosis, cerebral edema, and increased intracranial pressure, all of which are further aggravated by bacterial lysis from antibiotic therapy and the ensuing inflammatory response. Dexamethasone has been studied in children with meningitis to see if anti-inflammatory therapy can reduce brain injury and mortality. A meta-analysis of 18 randomized controlled trials in children found no effect of dexamethasone on mortality, but a subgroup analysis revealed significant reductions in hearing loss in children infected with Hib and children treated in high-income countries regardless of the infecting pathogen. The IDSA guidelines recommend the use of adjunctive IV dexamethasone (0.15 mg/kg/dose every 6 hours for 2 to 4 days) in children with Hib meningitis when it is initiated 10 to 20 minutes before or along with the first dose of antibiotics. Despite the lack of strong evidence to support its use in pneumococcal disease, dexamethasone use should be considered for children with pneumococcal meningitis after weighing the potential benefits and risks of therapy. Potential risks include adverse reactions, such as gastrointestinal effects and hypertension, and reduced antibiotic penetration to the site of infection, although studies have not confirmed this phenomenon. There is no evidence to support the use of dexamethasone in meningococcal meningitis.

In this patient, dexamethasone therapy could be considered because the causative pathogen is unknown (Gram stain of CSF was negative; culture is pending), and she has not received any vaccines. However, initiation of corticosteroid therapy is recommended with the first dose of antibiotics or 10 to 20 minutes before the first dose is given. This patient has not yet received parenteral antibiotics, but she received oral antibiotics prior to her presentation, which could interfere with the ability to identify the infecting pathogen. Her case could be considered a "partially treated" meningitis given the doses of amoxicillin prior to admission and data to support the protective effect of corticosteroids when given after antibiotic therapy are lacking. The IDSA guidelines recommend avoiding dexamethasone in patients who have already received antibiotics because it is unlikely to improve outcomes. Therefore, the use of dexamethasone for this patient is not recommended, and it should be discontinued.

4. Provide pertinent education for the parents regarding the prognosis and long-term outcomes for children with meningitis.

Response to treatment (improvement in vital signs and neurologic status) is usually evident within 48 hours of initiating antibiotic therapy. Therapeutic failure may occur because of inadequate antibiotic treatment, development of nosocomial infection, or development of suppurative complications such as subdural empyema. For children who are not improving or are worsening, it is recommended to repeat a lumbar puncture, particularly in cases of pneumococcal meningitis caused by penicillin-

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or cephalosporin-resistant strains and in those who received adjunctive dexamethasone therapy. Continued presence of infection after 48 hours of antibiotic therapy is associated with poor outcome. In these cases, changes in antibiotic selection, dosing, or duration may be necessary.

As discussed previously, meningitis can result in neurologic sequelae in almost 50% of children despite prompt and appropriate antibiotic therapy while mortality rates are much lower at approximately 2%. Certain factors, such as young age (<12 months), delayed presentation, presence of seizures or coma, need for vasopressors, and pneumococcal etiology, can increase the risk of poor outcomes. Prompt administration of antibiotics in children with a likely diagnosis of bacterial meningitis is recommended in order to minimize mortality, but little evidence exists to show that it significantly reduces morbidity. After the infection has been adequately treated, some neurologic deficits may improve or resolve over time, but subtle deficits can be initially overlooked and may affect survivors for years. Thus, it is important for children to undergo vision and hearing testing along with routine primary care visits and neurologic follow-up to monitor for complications.

5. Discuss the medication-related problem in this patient and provide a recommendation for its management.

The medication-related problem was the inappropriate amoxicillin dose for this patient's weight. High-dose amoxicillin (80 to 90 mg/kg/day divided twice daily) is the first-line treatment for children with AOM. This patient was diagnosed with AOM 3 days prior to admission and was prescribed amoxicillin 800 mg twice daily as an outpatient. This dose is too high for this patient's weight (167 mg/kg/day); an appropriate dose is 400 mg twice daily. The patient has evidence of middle ear effusion but no current evidence of inflammation; despite this, antibiotic therapy for AOM should be continued for a full treatment course as this case may represent a partially treated infection. The parenteral antibiotic therapy that is recommended for meningitis (ceftriaxone plus vancomycin) will also treat the AOM, so the amoxicillin can be discontinued. A 3-day course of ceftriaxone is an appropriate alternative treatment for AOM, and it is likely that this patient will receive 7 to 14 days of parenteral antibiotics for meningitis.

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CASE 10.1
Anemia | Level 1

Tracy M. Hagemann

1. What subjective and objective evidence supports the diagnosis of microcytic anemia?

SUBJECTIVE FINDINGS: The patient had an essentially normal presentation but was somewhat irritable and has had changes in sleep lately.

OBJECTIVE FINDINGS: Remarkable for decreased hemoglobin of 8.4 g/dL, decreased MCV of 59.4 fL, RDW 20% (increased), and RBC count decreased at 4.75 million/μL

The patient's diet consists solely of cow's milk increasing his risk for the development of this type of anemia.

2. Develop a nonpharmacologic and pharmacologic regimen for the treatment of iron-deficiency anemia in this patient.

Iron deficiency is characterized by microcytosis with an increased RDW. Untreated iron-deficiency anemia in infants and toddlers is associated with long-lasting delays in mental, motor, and behavioral functioning. Because this type of anemia is mild and this patient's history and laboratory values are consistent with iron deficiency, it is appropriate to treat empirically with oral iron therapy. The patient should have repeat laboratory testing performed 1 month after starting iron replacement. These labs should include hemoglobin, MCV, RBC count, and RDW. An increase in the hemoglobin level of more than 1 g/dL after 1 month of therapy confirms the diagnosis of iron-deficiency anemia. The patient should then be treated with iron replacement for an additional 3 to 4 months after the hemoglobin returns to normal values, in order to fully replenish the total body stores. If the hemoglobin does not increase after 1 month of therapy, further testing, including a complete blood count, peripheral blood smear, iron studies, and fecal occult blood testing should be performed.

Cow's milk has an iron content of about 0.5 mg/L and cannot meet the iron needs of infants and toddlers. It is estimated that a growing toddler requires about 11 mg/day of iron to maintain normal growth and development. This would mean that this child would need to drink 22 liters of cow's milk per day to meet his requirements. Because the suspected cause of the iron-deficiency anemia in this patient is a diet consisting of mostly cow's milk, the parents should be instructed to stop the patient's milk consumption entirely. He should

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be initiated on a diet with normal foods such as fruits, vegetables, meat, and iron-fortified cereals and other liquids, such as water and juice. With this patient's weight, juices should be limited to 1 cup per day. Milk may be added back into his diet once the anemia has started to improve but only with meals.

Ferrous sulfate is the most widely available product and care should be taken to ensure that the correct dose of elemental iron is administered. As this child is only 14-months old, an oral liquid product would be most appropriate. The recommended dose for treatment of anemia in pediatrics is 3 mg elemental iron/kg/day in one to two divided doses; this patient's weight is 12.2 kg, so he should receive 36.6 mg elemental iron per day (12.2 kg × 3 mg/day = 36.6 mg/day). To be most practical, it would be appropriate to round the patient's calculated dose to 36 mg/day.

Ferrous sulfate is available in an elixir, suspension, or oral drops. The elixir is available as 220 mg/5 mL (44 mg elemental iron/5 mL), while the oral drops are available as 75 mg/mL (15 mg elemental iron/mL). The suspension drops MyKidz Iron™ 10, is 75 mg/1.5 mL (15 mg elemental iron/1.5 mL) and is formulated for children with no dyes and is ethanol free. Other appropriate suspensions include Fer-In-Sol 75 mg/mL (15 mg elemental iron/1 mL) and Fer-iron 75 mg/mL (15 mg elemental iron/1 mL), although these both contain 0.2% alcohol. The pharmacist should provide an oral syringe that can measure the correct amount of liquid for this dose.

For this patient, who will require 36 mg of elemental iron/day, an appropriate regimen would be MyKidz Iron™ 10, 3.6 mL (36 mg elemental iron) once daily or 1.8 mL (18 mg elemental iron) orally twice daily. Once-daily dosing results in similar improvement as taking the iron twice daily, with no increase in adverse reactions. Using the other products of Fer-In-Sol or Fer-iron, the regimen would be 2.4 mL (36 mg elemental iron) orally once daily or 1.2 mL (18 mg elemental iron) orally twice daily.

3. Identify and propose a solution to the medication-related problem.

This patient has symptoms of seasonal allergies (e.g., sneezing and runny nose), and his mother has been administering diphenhydramine at bedtime for these symptoms. Diphenhydramine is a concern in this patient as he is only 14-months old, and diphenhydramine has a safety warning in children less than 2 years of age. The FDA notes that there are no approved OTC uses for cough and cold products in children less than 2 years of age, due to reported serious adverse reactions including death. Additionally, the parents of this child state that he has not been sleeping well for the past month. Diphenhydramine was started about a month ago and may cause paradoxical excitation in young children. The diphenhydramine should be discontinued immediately. Another product that may help for the treatment of seasonal allergic symptoms in children less than 2 years of age is cetirizine. Cetirizine dosing for children 12 to 23 months of age is 2.5 mg once daily and may be increased to 2.5 mg twice daily if needed for symptoms. Cetirizine is available as an oral syrup at 5 mg/5 mL, so this patient would initially require 2.5 mL (2.5 mg) once daily. Cetirizine is much less likely to cause excitation but may cause some drowsiness and is best to take at bedtime.

4. What is the patient's current weight classification? What is the desired weight for this patient?

This patient is at the 97 percentile for weight and height per the Centers for Disease Control and Prevention/National Center for Health Statistics. While body mass index (BMI) is only appropriate for children older than 2 years, weight for length percentile is the appropriate method for assessing risk for under or overweight. Percentiles less than 5 indicate underweight, percentiles between 5 and less than 95 indicate healthy weight, and percentiles over 95 indicate overweight. A desired weight for length percentile for this patient would be between 5 and 95 percentile, or with a length of 78 cm, a weight below 12 kg. At the extremes (percentiles greater than 97 or less than the 3), small

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differences in percentiles can represent important differences in weight/height.

5. Devise the most pertinent patient education for the caregiver regarding both pharmacologic and nonpharmacologic therapy.

The parents and other caregivers of this child should ensure that the patient receives the iron product daily as prescribed. As the liquid iron supplements may be difficult for a toddler to take, due to the taste of the medication, the parents may administer the medication with orange juice: this will assist with absorption of the medication as well as mask the taste. Parents should be advised that the use of iron-containing products may cause the patient's stools or urine to have a black or dark colored appearance and that this is normal. The patient may have symptoms of stomach pain, nausea, or constipation. The iron may be administered with food to reduce stomach pain and nausea but should not be given with milk or milk-containing foods, such as pudding or cereal. Iron supplementation should not be administered with antacids.

While this patient is above average for weight, as he resumes a normal diet, height and weight should be monitored for any changes. No specific therapy is needed at this time for his weight. Parents should limit excessive snacking and limit consumption of juice to no more than 1 cup per day. Activity during wakening hours should be encouraged.

The patient's eczema appears well controlled, but he does have dry patches on his lateral thigh. The parents should bathe him regularly, with baths preferred over showers. Baths should be warm, not hot, and should last no longer than 10 minutes. The use of soap should be minimized and bubble bath and other bath additives should be avoided, because they can be irritating to the skin and worsen the eczema. Cleansers such as Cetaphil and CeraVe may be used to wash the diaper area and any other areas that appear dirty. Rough washcloths, loofas, and any skin scrubbers should also be avoided. A moisturizer should be applied to damp skin immediately after bathing. Moisturizers should be applied twice daily in a thick layer.

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CASE 10.2
Sickle Cell Disease | Level 2

Tracy M. Hagemann

1. What subjective and objective evidence supports the diagnosis of a sickle cell pain crisis and of acute chest syndrome?

PAIN CRISIS

SUBJECTIVE FINDINGS: Self-reported pain started as 5/10 on VAS and increased to 10/10; pain described as sharp, throbbing, worse with movement and with deep breaths; home pain medications did not help decrease pain; in moderate distress

OBJECTIVE FINDINGS: Increased pulse, increased respiratory rate

ACUTE CHEST SYNDROME

SUBJECTIVE FINDINGS: Described chest pain on left side, worse with deep breaths; reported nonproductive cough for 4 days

OBJECTIVE FINDINGS: CXR shows no new infiltrates, but infiltrates may take a day or two to manifest; chest auscultation demonstrates decreased aeration on left; decreased oxygenation per pulse oximeter reading; increased respiratory rate; increased temperature

2. Develop a pharmacologic regimen for the treatment of the pain crisis and the suspected acute chest syndrome in this patient.

Patients with pain that cannot be adequately controlled on home medications, as well as any patient with SCD who has chest pain or signs and symptoms of acute chest syndrome, should be admitted for inpatient therapy. Goals of therapy for the treatment of pain crisis are to relieve pain (at least back to their baseline), increase hemoglobin levels if decreased, maintain adequate hydration, and prevent complications from the use of opioids (e.g., itching, nausea, sedation, respiratory depression, and constipation). It is unrealistic to expect patients to have no pain; because this is a chronic disease some level of pain is almost always present, but pain should be controlled as much as possible for patients to be able to function well and adequately perform activities of daily living.

Goals of therapy for acute chest syndrome include maintaining oxygen saturations at or above 92% on room air; treating bacterial infections if present (or at least treat empirically until resolution of symptoms of infection such as fever, increased WBC count, chest pain, and cough); resolving symptoms such as chest pain and increased respiratory rate;

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increasing hemoglobin levels if decreased; and maintaining adequate hydration.

PAIN CRISIS

The patient should receive IV fluids at 1 to 1.5 times maintenance for a total of 2,380 mL/day or 100 mL/hr. She also has suspected acute chest syndrome, so IV fluids should be limited to maintenance rates to avoid fluid overload. Because of a hemoglobin level of 6.5 g/dL, she should be transfused with pRBCs 640 mL (10 mL/kg) over 4 hours. All patients with SCD who require transfusions should be transfused with leukocyte-poor, antigen-matched blood to reduce the frequency of transfusion reactions and the development of antibodies. Generally, transfusion should only be administered if the hemoglobin level is 1.5 times below baseline or less than 7 g/dL, and the patient requires oxygen supplementation.

The severe pain the patient is experiencing, self-rated as 10/10 and not responding to oral pain medications, is being treated with a morphine PCA with a dose of 1.3 mg (0.02 mg/kg) IV every 10 minutes with a basal of 3.8 mg (0.02 to 0.06 mg/kg/hr). The use of the higher basal dosing is appropriate given her uncontrolled pain and pain ratings of 10/10. Alternately, she could receive a continuous infusion of morphine at 2 mg/hr (0.02 to 0.05 mg/kg/hr) titrated to effect, or intermittent IV dosing of 5 mg (0.05 to 0.15 mg/kg) IV every 3 to 4 hours prn. In this patient's case, the use of the PCA with basal dosing will give her more control over her intermittent dosing and may result in an overall lower opioid dose administered, which may decrease adverse reactions from the morphine such as constipation, itching, drowsiness, respiratory depression, and sedation. She should also receive an NSAID, such as ketorolac 30 mg (0.5 mg/kg) IV every 6 hours scheduled for at least 48 hours and up to 5 days. Ketorolac should not be used for longer than 5 continuous days due to increased incidence of adverse reactions such as gastrointestinal bleeding. After 5 days of ketorolac, she may be changed to ibuprofen 600 mg (10 mg/kg rounded to the nearest tablet size) po every 6 to 8 hours until the pain crisis has resolved.

The morphine therapy may cause constipation; therefore, a stool softener should be initiated prophylactically. Docusate 100 mg po tid (5 mg/kg/day divided in one to four doses) was ordered at admission. Consider changing to a laxative, such as polyethylene glycol 3350 (MiraLAX®) 17 g mixed in water or juice once daily, if the patient complains of constipation. Opioid adverse reactions also include itching, which may be treated initially with diphenhydramine 25 mg (1 mg/kg/dose) IV or po every 6 hours (max 25 to 50 mg/dose) and if itching remains uncontrolled a low-dose naloxone drip of 16 mcg/hr IV infusion (initiate at 0.25 and increase to no more than 1 mcg/kg/hr if necessary), concomitant with the morphine PCA or continuous infusion should be considered. If she develops nausea or vomiting from the use of the opioid, ondansetron 4 mg (0.1 mg/kg/dose with max dose of 4 mg) IV or po every 8 hours, or promethazine 16 mg (0.25 mg/kg) IV or po every 6 hours for a maximal dose of 25 mg/dose, may be useful. Ondansetron should be initiated first to prevent nausea and vomiting; it generally has fewer adverse reactions such as sedation, than promethazine; however, once the patient has started vomiting, the promethazine should be started because ondansetron is not as effective for treating vomiting as promethazine. If the adverse reactions of the morphine become unmanageable, it may be helpful to change to a different opioid such as hydromorphone.

ACUTE CHEST SYNDROME

Fluid therapy should be monitored closely; while necessary, it is important to avoid fluid overload, which may lead to pulmonary edema and increased respiratory distress. In light of this patient having both acute chest syndrome suspected as well as a known pain crisis, IV fluids should be kept at no more than daily maintenance requirements. Close attention should be paid to her daily intake and output to ensure fluid balance. Oxygen per nasal cannula should be started to keep the oxygen saturation at greater than 92%. Because her hemoglobin is low (6.2 g/dL), she should receive a transfusion of 640 mL (10 mL/kg) of pRBCs. Her hemoglobin should be monitored, and a goal of her normal baseline hemoglobin should be made; the goal is to get the patient back to her base-

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line within 24 to 48 hours. This should improve her oxygenation. Antimicrobial therapy with ceftriaxone 2 g IV (50 mg/kg/day to maximum dose 2 g) every 24 hours and azithromycin 250 mg po daily should be initiated to cover for *S. pneumoniae, S. aureus, H. flu,* and *Mycoplasma* spp. If the patient has had a history of invasive *S. pneumococcus* infections in the past, it may be prudent to also add empiric coverage with vancomycin 15 mg/kg IV every 8 hours. The patient should be encouraged to use incentive spirometry every 2 hours to prevent atelectasis and improve lung expansion. She should be closely monitored for hypoventilation from the opioids used for pain management. Adverse reactions from the use of antibiotics may include rash, diarrhea, nausea, vomiting, and abdominal pain.

Her other home medications, such as folic acid and hydroxyurea, should be continued during her hospital stay.

3. Assess the pharmacologic therapy for each of the patient's problems.

PAIN CRISIS

The initial therapy is appropriate, with the use of the morphine PCA and the IV ketorolac, as well as the transfusion and IV fluids. She does not have an order written to treat or prevent potential nausea/vomiting, so addition of either ondansetron or promethazine for prn usage may be prudent. Her pain should be assessed and documented at least every 4 hours using a VAS tool, as well as shift totals for the number of intermittent doses of morphine received via PCA. For as long as she is receiving opioids via PCA, she should be on continuous pulse-oximeter to monitor her oxygen saturations. All vital signs (respiratory rate, pulse, temperature, and blood pressure as well as the appearance of opioid-induced itching) should be assessed each shift or at least every 4 hours. If she does not produce a stool within 24 hours, docusate may not be enough to counteract the constipating effects of the opioids; therefore, a laxative such as polyethylene glycol 3350 may be necessary. Ambulation with assistance should be encouraged, if possible, to assist with relief of constipation and to improve airway expansion.

ACUTE CHEST SYNDROME (SUSPECTED)

The initial therapy is appropriate. Her IV fluids are running at approximately 100% maintenance, and it may be appropriate to decrease to 75% of maintenance if she develops signs or symptoms of worsening respiratory function, such as increased oxygen needs, increased respiratory rate, and decreased breath sounds on auscultation. Ceftriaxone and azithromycin are appropriate first-line antimicrobials for a suspected acute chest syndrome, especially because cultures are pending at this time. The pRBC transfusion should help to increase oxygenation and improve oxygen saturations.

ANEMIA

Because the patient is anemic, it is appropriate to receive a pRBC transfusion to help increase her hemoglobin back to her baseline value. Hyperviscosity may occur if her hemoglobin concentration is increased to greater than 10 to 11 g/dL, so the goal for her should be her usual baseline hemoglobin concentration. Generally, the hemoglobin should be checked about 1 hour after the transfusion is completed. Alloimmunization associated with frequent transfusions can be minimized through the use of leukocyte-reduced RBCs or HLA-matched units.

4. Devise the most pertinent education for the patient and caregiver regarding both pharmacologic and nonpharmacologic therapy.

The patient has morphine via PCA and should understand that the pump will continue to deliver a small dose around the clock, but she may use the "button" to deliver additional doses when she has increased pain or anticipates pain (e.g., needing to cough, getting out of bed). She should press the button regardless of when she last received an intermittent dose and should not watch the clock and only push the button every 20 minutes when she is "due" for another dose. She should inform the nurse if there is a sudden worsening of pain or increased difficulty in breathing. She should use her incentive spirometry every 2 hours and keep her oxygen cannula in her nostrils unless instructed to remove it. She should be encouraged to continue to drink fluids and record her

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urine output (or inform her nurse that she has urinated) so that there is an accurate measurement of her ins/outs.

She should be aware of the most common adverse reactions associated with her inpatient therapy, such as opioid-induced itching, sedation, respiratory depression, constipation, and nausea/vomiting. Because the diphenhydramine order for itching is written as prn, if she starts itching, she should notify her nurse and request a dose. If she has not had a bowel movement within 1 to 2 days after being started on the docusate or starts having stomach pains or other symptoms of constipation, she may need to be changed to a laxative as described above. She does not have an as-needed order written for treating or preventing nausea or vomiting, and if she begins to feel nauseated or vomits, she should let her nurse know quickly. She should keep her pulse-oximeter probe on to help monitor her oxygen saturations; this monitor will alarm if her oxygenation falls below 92%. The antibiotics that she is receiving may cause nausea/vomiting, abdominal pain, or diarrhea; if any of these symptoms occur, she should notify her nurse. She should be counseled that she is receiving an NSAID, ketorolac, and it may increase her risk of bleeding, especially gastrointestinal bleeding; if she starts to have abdominal pain, gastrointestinal fullness, heartburn, nausea, or vomiting, she should notify her nurse immediately. Ketorolac and other NSAIDs may also cause renal dysfunction, and she should be informed that her renal function will be monitored, including BUN/creatinine and daily intake and output for fluctuations.

5. Why would this patient be using hydroxyurea? Assess the dose and describe how you should monitor her use of this drug.

Hydroxyurea is a ribonucleotide reductase inhibitor that has been shown to increase fetal hemoglobin (HbF) levels. Red blood cells that contain HbF are less likely to sickle, because HbF binds oxygen more tightly than hemoglobin A. Most adult RBCs contain less than 1% HbF. Hydroxyurea has been shown in both pediatric and adult studies to reduce the frequency of hospitalizations and the incidence of pain crises, acute chest syndrome, and the need for blood transfusions by almost 50% in those with moderate-to-severe SCD. Hydroxyurea is recommended as an option in children and adolescents with moderate-to-severe SCD and should be considered if the patient has any history of acute chest syndrome, other severe vaso-occlusive events (generally more than three in 1 year), or severe symptomatic anemia. Because this patient has a history of multiple hospitalizations for pain crises, her disease would be considered moderately severe, and a trial of hydroxyurea would be warranted.

She is currently receiving hydroxyurea 1,000 mg po bid (31 mg/kg/day), and she has been on this dose for almost 1 year. Doses should be initiated at 10 to 15 mg/kg daily as a single dose and may be increased after 8 to 12 weeks if tolerated. Doses can be increased by 5 mg/kg/day up to 35 mg/kg/day. Patients should be monitored for efficacy and toxicity because not all patients will respond equally to the drug. Mean corpuscular volume (MCV) will increase as the HbF concentration increases and are a surrogate marker for efficacy. If the MCV does not increase, the patient's bone marrow may be unable to respond, the dose may be inadequate, or the patient may be nonadherent to the medication. HbF levels can also be monitored with a goal of increasing the HbF to 15% to 20%. Complete blood counts should be monitored every 2 weeks during titration and then every 4 to 6 weeks once the dose is stabilized. Therapy should be temporarily discontinued if signs of toxicity occur: hemoglobin less than 5 g/dL, decreased absolute neutrophil count (ANC), decreased platelets, or if the reticulocytes are decreased when the hemoglobin is less than 9 g/dL. Patient should be monitored for decreased renal and hepatic function. All patients receiving hydroxyurea should also receive folic acid supplementation because folate deficiency may be masked by the use of hydroxyurea.

6. Identify and propose a solution to a medication-related problem.

This patient completed the childhood immunization series that was appropriate at the

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time (15 years ago) and has been adherent to receiving an annual dose of influenza vaccine. Children with SCD develop impaired splenic function during childhood which increases their risk of infection with encapsulated bacteria (e.g., *S. pneumoniae, H. influenza, Salmonella* spp.); therefore, all children with SCD should receive a dose of 23-valent polysaccharide vaccine at age 2 years, with a second dose given at least 5 years after the initial dose. The 23-valent polysaccharide pneumococcal vaccine contains the most common isolates seen in older children and adults; however, there is still a risk for pneumococcal infections in this population. The 13-valent conjugate pneumococcal vaccine is now also recommended for any patient with SCD who has not already received it. This patient has not received the 13-valent conjugate vaccine in the past, so she should receive one dose before discharge from the hospital. Because she only received one dose of the 23-valent polysaccharide vaccine and it has been more than 5 years since her first dose, she also needs a dose of this vaccine. However, according to the CDC, if the 13-valent vaccine is administered first, she will need to wait 8 weeks to be given a dose of the 23-valent polysaccharide vaccine; this can be done at a follow-up outpatient appointment. In addition to vaccination for pneumococcal disease, she is at risk for meningococcal disease due to her functional asplenia. She should be vaccinated with the meningococcal conjugate vaccine (Menactra®) and should receive two doses, 2 months apart. If it has been at least 2 years since her last tetanus-diphtheria vaccination, she should also receive a one-time dose of Tdap.

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CASE 10.3
Anticoagulation | Level 3

Brady S. Moffett

1. What is the subjective and objective evidence for the diagnosis of arterial thrombosis in this patient?

SUBJECTIVE FINDINGS: Appearance of decreased perfusion (grey, sluggish capillary refill, cold) and distal pulses in that hand were weak.

OBJECTIVE FINDINGS: Doppler ultrasound indicative of decreased perfusion, increased capillary refill time in fingers, weak distal pulses, and cool temperature of fingers.

Risk factors for development of an arterial thrombus include sepsis, underlying coagulopathy, and the presence of arterial catheter.

2. Devise a pharmacologic regimen for the treatment and prophylaxis of arterial thrombosis in a critically ill patient.

The goal for anticoagulation therapy in this patient is to restore perfusion to the affected limb without any major bleeding complications. The patient will have achieved this if the color, temperature, and pulses to the limb have been restored without any serious adverse reactions related to anticoagulation therapy.

Prior to initiation of anticoagulation therapy, any agent responsible for the generation of the arterial thrombus—in this case, the arterial catheter—should be removed if possible. Placement of monitoring catheters in other sites may be an option if they are still deemed necessary.

Unfractionated heparin (UFH) is a suitable first choice of pharmacotherapy because this patient is experiencing signs of decreased perfusion to the limb. UFH can be titrated every 6 hours to attain a therapeutic activated partial thromboplastin time (aPTT) and/or factor Xa concentration. The factor Xa may be a more sensitive test to determine the efficacy of UFH therapy because it is not as easily influenced by disease states. Low molecular weight heparin (LMWH) such as enoxaparin can be used, but because these agents are dosed every 12 hours, the time for titration to a therapeutic concentration may be lengthy. Case series of the use of enoxaparin in arterial thrombus with success have been published.

Tissue plasminogen activator (tPA) can be used to lyse the thrombus if the patient were at immediate risk of losing the limb from the thrombosis. The risk of bleeding is high with use of tPA and should be balanced with the benefit of using tPA. Those at risk of bleeding

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include premature infants, patients who have recently undergone a surgical procedure, and patients with baseline bleeding disorders. UFH therapy should be continued during tPA infusion to prevent extension of the thrombus. The risk of bleeding in this patient is high because they are coagulopathic, at baseline, and have reduced drug clearance due to decreased kidney function. The use of tPA should only occur if UFH therapy does not improve perfusion to affected limb within the first 12 hours of therapy or if the perfusion status of the limb decreases significantly while receiving UFH therapy.

For the patient in this case, the use of UFH should occur as soon as possible. Recommended doses of IV UFH should be initiated at 20 units/kg/hr as a continuous infusion, preceded by an IV bolus dose of 75 units/kg (maximum of 5,000 units). Therapy with tPA, should it be required, typically can be initiated at 0.1 mg/kg/hr as a continuous infusion, increasing each hour to a maximum of 0.6 mg/kg/hr over a 6-hour period. The tPA infusion can be discontinued prior to the 6-hour infusion period if perfusion to the affected limb has markedly improved.

3. Determine the appropriate monitoring for the use of anticoagulation therapy in a critically ill patient with an arterial thrombosis.

Standard laboratory monitoring for a patient receiving UFH is the aPTT. A factor Xa assay can be useful in patients with underlying coagulopathy because baseline coagulopathy can affect the aPTT, making the results unreliable in assessing UFH effect. An institution's therapeutic aPTT range is based on a factor Xa concentration of 0.3 to 0.7 units/mL. The factor Xa assay is not readily available at every institution and is typically a resource intensive test, which is not as rapidly available as an aPTT. In this patient, the use of a factor Xa assay would be useful because the patient's underlying disease process (i.e., sepsis) and disease-related coagulopathy would likely alter the aPTT. Initial monitoring would include an aPTT every 6 hours, starting with a baseline aPTT prior to initiation of UFH therapy. Most institutions have a goal aPTT in the 60- to 90-second range for a therapeutic UFH goal, but institutions will vary and the laboratory should be consulted for the specific range. If a factor Xa assay is available and the aPTT appears to be an unreliable measure of UFH effect, factor Xa monitoring should occur at least every 12 hours, with proportional adjustment in UFH therapy to achieve a goal of 0.3 to 0.7 units/mL.

To help determine the validity of an aPTT value in a patient with a disease state that can result in coagulopathy, the use of a heparinase enzyme with the aPTT (HepZyme aPTT) will neutralize the effects of UFH and allow the clinician to investigate the patient's baseline aPTT to assess for coagulopathy. If coagulopathy is present, the HepZyme aPTT will be altered and the results of the aPTT will likely not represent true anticoagulation effect of UFH. In a patient with an altered baseline aPTT, as identified by using the HepZyme aPTT, the factor Xa assay should be used. In situations where advanced coagulation testing (factor Xa assay, HepZyme aPTT) is unavailable, the clinician should be aware of the influences of patient disease processes on laboratory monitoring parameters for UFH.

Monitoring should be continued to assess for bleeding complications, such as a drop in platelets, hemoglobin, or hematocrit, as well as more obvious signs of bleeding, such as oozing from IV cannula sites or bleeding from mucous membranes. Laboratory monitoring for anticoagulation should occur frequently, every 4 to 6 hours in this particular patient, due to their high risk of bleeding. Bleeding from IV cannula sites and mucous membranes can be assessed on a continual basis in the intensive care unit. Similarly, neurologic status should be monitored continuously to assess for intracranial or intracerebral bleeding. Sudden or dramatic changes in neurologic status, such as a seizure or change in neurologic exam, would warrant the use of advance imaging techniques for occult bleeding.

Platelet monitoring during UFH therapy is necessary to detect heparin-induced thrombocytopenia (HIT), a rare but devastating complication in pediatric patients receiving UFH. HIT Type I is characterized by a slow decrease in platelets and is due to platelet consumption or

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bleeding and is not typically associated with adverse outcomes. HIT Type II is an immunologic response to UFH characterized by a greater than 50% decrease in platelet count within 72 hours of UFH initiation, thrombosis, and bleeding. Patients with HIT Type II should have all UFH therapy discontinued, including UFH in monitoring catheters, and anticoagulation should be switched to a direct thrombin inhibitor, such as argatroban.

For this patient, baseline assessment of clotting system function should occur (prothrombin time, aPTT, antithrombin III, platelets) prior to initiation of UFH therapy. The risk of underlying coagulopathy is present because this patient has sepsis. A factor Xa, aPTT, and HepZyme aPTT should be drawn at 6 hours after UFH therapy has been initiated. If the hospital therapeutic range for aPTT on UFH therapy does not correlate to a factor Xa concentration of 0.3 to 0.7 units/mL, the aPTT is unreliable for assessment of UFH therapeutic effect and the factor Xa concentration should be followed. It should be noted that the patient in this case has an elevated aPTT at baseline (43 seconds), and the factor Xa assay will likely prove to be the methodology for assessment of UFH therapy.

4. Determine appropriate dosing of medications in a patient with sepsis and acute kidney injury.

Prior to diagnosis of acute kidney injury (AKI), the patient's urine output should be quantified and glomerular filtration rate (GFR) should be estimated by using the original Schwartz equation. The bedside Schwartz equation, another tool for estimating GFR, was validated in patients with chronic kidney disease and may be less accurate than the original Schwartz equation. However, it is important to note that these equations estimate GFR and accuracy of the calculations may be low in a critically ill patient with rapidly changing kidney function. The diagnosis of AKI is made by using the pediatric modified RIFLE criteria (pRIFLE). The estimated GFR in the patient is approximately 34 mL/min/1.73 m^2 calculated as [height (cm) × adjustment factor (k)]/serum creatinine (mg/dL)]. The urine output is 0.2 mL/kg/hr. This patient should be categorized as having grade "F" (failure) of AKI.

The AKI is likely secondary to hypoperfusion of the kidney from septic shock. Because the estimated GFR and urine output are likely diminished from baseline, renally filtered medications should be adjusted for kidney dysfunction.

Piperacillin/tazobactam is dosed based on piperacillin content and should be reduced to 50 mg/kg/dose (1.75 mg of piperacillin—decreased from 100 mg/kg/dose of piperacillin content) every 6 hours. Ranitidine should also be decreased, based on estimated GFR, to 1 mg/kg/dose (35 mg) every 12 hours.

Other medications are titrated based on patient response. Dopamine is titrated based on patient perfusion status—assessed by capillary refill time, trends in serum lactate, and peripheral temperatures as compared to a core temperature—while fentanyl and midazolam is titrated based on the analgesia and sedation that is required. For sedative and analgesic medications, continual assessment of patient requirements is necessary, and attempts at decreasing the amount of medication required to achieve analgesic and sedation goals should be frequent.

Vancomycin doses and schedules should be adjusted based on attainment of therapeutic serum concentrations. For this patient, a goal vancomycin trough value of 15 to 20 mg/L would be appropriate. Because the patient's GFR and urine output are diminished, the patient will likely require a dosing interval that is greater than every 6 hours. Serum sampling prior to the fourth dose is standard method for assessing attainment of goal serum concentrations. Alternatively, if this patient was yet to receive vancomycin therapy, a strategy using first-dose pharmacokinetics and sampling multiple serum concentrations after this first dose (i.e., at 2 and 8 hours after the dose) would allow for earlier determination of vancomycin pharmacokinetics and dosing strategies, thus optimizing efficacy and minimizing toxicity. Based on this patient's renal function, a single dose of vancomycin 525 mg (15 mg/kg/dose) would be appropriate with serum concentrations monitoring at 2 and 8 hours, initially, to

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perform first-dose pharmacokinetic calculations. Repeat serum concentrations should be drawn as necessary to accurately determine pharmacokinetic parameters and achieve goal serum trough concentrations of 15 to 20 mg/L.

Initiation of UFH in this patient is complicated by the sudden decrease in kidney function. UFH therapy is affected by decreased kidney function, and the risk of accumulation of UFH—and subsequent bleeding events—is high. The risk of using a bolus and subsequent bleeding should be weighed with the urgency of attainment of therapeutic concentrations and potential of limb loss. No empiric recommendations for decreasing doses prior to initiation of therapy are currently present, but clinicians should be cognizant of the potential for adverse reactions. The use of a bolus dose may put the patient at an increased risk of bleeding; therefore, initiation of UFH continuous infusion at 20 units/kg/hr with baseline and every-6-hour monitoring of aPTT and, if available, factor Xa, would be recommended. Bolus dosing of UFH can be used if the patient does not achieve therapeutic anticoagulation goals in the first 12 hours of therapy.

5. Develop a strategy for continuation of outpatient anticoagulation therapy and to overcome common medication-related problems in the continuation of outpatient therapy.

Therapy for the acute treatment of an arterial thrombosis has been UFH with the potential use of tPA in severe cases. After UFH therapy, the patient should be maintained on a longer-term regimen of anticoagulation, approximately 6 to 9 months, which should be initiated prior to discharge from the hospital. UFH therapy is not an ideal therapy for outpatient use because it is often given as continuous infusion through IV access and requires daily, if not more frequent, monitoring. Two options for use include warfarin and enoxaparin. Warfarin is an oral agent taken once daily but can be subject to drug and food interactions, which can result in bleeding or suboptimal therapy. Monitoring for warfarin includes the international normalized ratio (INR), which should be checked weekly until the patient is stable, potentially moving out to a once-monthly regimen. Doses of warfarin can vary widely, and frequent changes in dose may be common. Enoxaparin is less susceptible to drug–drug and drug–food interactions but is given twice daily as a subcutaneous injection, which may be painful and cause bruising. Monitoring of an antifactor Xa with enoxaparin can occur less frequently than INR monitoring with warfarin. The dose of enoxaparin in this patient is 1 mg/kg/dose (35 mg) subcutaneously twice daily. Enoxaparin is available as prefilled syringes in 30 and 40 mg doses; other doses would need to be prepared by a specialized outpatient pharmacy. The patient could use the 40 mg prefilled syringe and "squirt out" the extra drug, but this is not recommended because it may lead to dosing errors. The patient is also a teenager, and compliance may be difficult, regardless of anticoagulation therapy used. Overall, assessment of the patient's preference will be instrumental in deciding the choice of therapy. If subcutaneous injections are not a concern, the use of a 30 mg prefilled syringe twice daily and following antifactor Xa concentrations would be a reasonable initial plan. If antifactor Xa concentrations are suboptimal, the 40 mg prefilled syringe could be an option. As always, if the resources are available to provide specialty pharmacy services, this would be an ideal option.

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APPENDIXES

APPENDIX 1

DATA COLLECTION SHEET..... 254

APPENDIX 2

COMMON MEDICAL TERMINOLOGY AND ABBREVIATIONS IN PEDIATRIC PRACTICE 257

APPENDIX 3

"DO NOT USE" ABBREVIATIONS LIST..... 260

APPENDIX 4

SOAPE OUTLINE—
Multiple Days or Visits 261

APPENDIX 5

SOAPE OUTLINE—
Single Day or Visit 265

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APPENDIX 1: DATA COLLECTION SHEET

Patient name	**Caregiver name/relationship**
Patient ID#	**DOB**
Patient MR#	**Gender**
Room#	**Height/weight**
Admission date/initial visit	**Insurance/ethnicity**

Allergies/Intolerances/ADRs		**CC/HPI**
☐ No known allergies/ADRs		
☐ Not known/inadequate information		
Drug/food	**Reaction**	

FHx/SHx	**PMH**		

Immunizations

Vitals/Labs/Dx Tests

Date										
Weight										
Vitals										
Temp										
BP										
HR										
RR										
Chemistry										
Na										
K										
Cl										
CO_2										
BUN										
SCr										
CrCl										
Drug levels										

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APPENDIX 1 (cont'd)

CBC/Coags										
WBC										
Plt										
Hgb										
HCT										
aPTT										
PT/INR										
Cultures										
Other										
Dx tests										
Eating? Tube feeds?										
Formula Amount										

Physical Exam (pertinent findings from H&P)

Medications

Healthcare Problem List	**Problem**	**Current Pharmacotherapy (dose, formulation, route, frequency)**	**Assessment**

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APPENDIX 1 (cont'd)

Consultations:

Date	Progress Notes (e.g., physical exam findings, laboratory and diagnostic test results)

Plan

Date	Health-Related Problem	New Treatment Plan	Goals of Therapy	Therapeutic Endpoint	Monitoring Plan

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APPENDIX 2: COMMON MEDICAL TERMINOLOGY AND ABBREVIATIONS IN PEDIATRIC PRACTICE

Abbreviation	Medical Terminology
AACAP	American Academy of Child and Adolescent Psychiatry
ACE	Angiotensin-converting enzyme
ACTH	Adrenocorticotropic hormone
ADHD	Attention deficit hyperactivity disorder
AKI	Acute kidney injury
ALL	Acute lymphoblastic leukemia
ALP	Alkaline phosphatase
ALT	Alanine transaminase
ANC	Absolute neutrophil count
AOM	Acute otitis media
AOP	Apnea of prematurity
APGAR	Appearance, Pulse, Grimace, Activity, Respiration
aPPT	Activated partial thromboplastin time
AST	Aspartate aminotransferase
ATC	Around the clock
ATN	Acute tubular necrosis
AU	Each ear; both ears
AUC	Area under the plasma drug concentration-time curve
BAL	Bronchoalveolar lavage
BE	Base excess
bid	Twice daily
BMI	Body mass index
BP	Blood pressure
BPD	Bronchopulmonary dysplasia
BPM	Beats per minute
BS	Bowel sounds
BUN	Blood urea nitrogen
BW	Birth weight; body weight
CA-MRSA	Community-associated methicillin-resistant *Staphylococcus aureus*
CAP	Community-acquired pneumonia
cART	Combination antiretroviral therapy
CBC	Complete blood count
CD	Crohn disease
CDC	Centers for Disease Control and Prevention
CF	Cystic fibrosis
CFRD	Cystic fibrosis–related diabetes
CMP	Comprehensive metabolic panel
CNS	Central nervous system
CO_2	Carbon dioxide
CPR	Cardiopulmonary resuscitation
CPAP	Continuous positive airway pressure
CRP	C-reactive protein
CT	Computerized tomography
CTA	Clear to auscultation

Abbreviation	Medical Terminology
CTABL	Clear to auscultation bilaterally
CXR	Chest x-ray
DASH	Dietary approaches to stop hypertension
DKA	Diabetic ketoacidosis
DRE	Digital rectal exam
DNA	Deoxyribonucleic acid
DS	Double strength
DTaP	Diphtheria, tetanus, and pertussis (vaccine)
DTR	Deep tendon reflexes
DVT	Deep vein thrombosis
ECG	Electrocardiogram
ECHO	Echocardiogram
ECL	Electrochemiluminescent immunoassay
ED	Emergency department
EEG	Electroencephalogram
ELBW	Extremely low birth weight
ENT	Ear, nose, and throat
EOM(I)	Extraocular muscles (intact)
EPS	Extrapyramidal symptoms
ESR	Erythrocyte sedimentation rate
ESRD	End-stage renal disease
ET	Endotracheal
ETT	Endotracheal tube
FDA	Food and Drug Administration
FLACC	Face, legs, activity, cry, and consolability
FSH	Follicle-stimulating hormone
FTT	Failure to thrive
G-tube	Gastrostomy tube
GABA	Gamma-aminobutyric acid
GBS	Group B *Streptococcus*
GCS	Glasgow coma scale
GERD	Gastroesophageal reflux disease
GFB	Growth factor-binding
GFR	Glomerular filtration rate
GH	Growth hormone
GI	Gastrointestinal
GLAD-PC	Guidelines for Adolescent Depression in Primary Care
GMFCS	Gross Motor Function Classification System
G1P1	Gravida 1, para 1
GPC	Gram-positive cocci
GU	Gastric ulcer; genitourinary
HA	Headache
HbA_{1C}	Glycosylated hemoglobin (used as a measure of diabetes control)
HbF	Fetal hemoglobin
HC	Head circumference
HCO_3	Bicarbonate

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APPENDIX 2 (cont'd)

Abbreviation	Medical Terminology
HDL	High-density lipoprotein
HEENT	Head, eyes, ears, nose, throat
HFA	Hydrofluoroalkane
Hg	Hemoglobin
Hib	*Haemophilus influenzae* type b
HLA	Human leukocyte antigen
HPA	Hypothalamic-pituitary-adrenal
HPI	History of present illness
HIT	Heparin-induced thrombocytopenia
hr	Hour
HR	Heart rate
hs	At bedtime (from Latin *hora somni*)
HSM	Hepatosplenomegaly
Ht	Height
HTN	Hypertension
Hz	Hertz
IBD	Inflammatory bowel disease
iCa	Ionized calcium
ICU	Intensive care unit
IDMS	Isotope dilution mass spectrometry
IGF-1	Insulin-like growth factor 1
IGF-2	Insulin-like growth factor 2
IGFBP-3	Insulin-like growth factor–binding protein 3
IIV	Inactivated influenza vaccine
IM	Intramuscular
INR	International normalized ratio
I/O	Input/output ("ins" and "outs")
IPV	Inactivated polio vaccine
IQ	Intelligence quotient
IT	Intrathecal
IV	Intravenous
IVF	Intravenous fluid
IVIG	Intravenous immune globulin
LBW	Low birth weight
LDA	Lines, drains, and airway
LDL	Low-density lipoprotein
LFT	Liver function test
LGA	Large for gestational age
LGID	Low glycemic index diet
LGIT	Low glycemic index treatment
LH	Luteinizing hormone
LLSB	Lower left sternal border
LMP	Last menstrual period
LMWH	Low molecular weight heparin
LN	Lymph node
LP	Lumbar puncture
LUL	Left upper lobe

Abbreviation	Medical Terminology
LVH	Left ventricular hypertrophy
MAP	Mean arterial pressure
MAR	Medication administration record
MCH	Mean corpuscular hemoglobin
MCHC	Mean corpuscular hemoglobin concentration
MCV	Mean corpuscular volume
MDD	Major depressive disorder
MDI	Metered-dose inhaler
MGF	Maternal grandfather
MGM	Maternal grandmother
MIC	Minimal inhibitory concentration
min	Minute
MRI	Magnetic resonance imaging
MIVF	Maintenance intravenous fluids
MM	Mucous membrane
MMR	Measles, mumps, and rubella (vaccine)
mo	Month
MPV	Mean platelet volume
m/r/g	Murmurs/rubs/gallops
MRSA	Methicillin-resistant *Staphylococcus aureus*
MS	Mental status; mitral stenosis; muscle strength
msec	Millisecond
MSSA	Methicillin-susceptible *Staphylococcus aureus*
MVI	Multivitamin infusion
NAFLD	Nonalcoholic fatty liver disease
NC	Nasal cannula
NG	Nasogastric
NGTD	No growth to date
NICU	Neonatal intensive care unit
NKDA	No known drug allergies
NPO	Nothing by mouth (from Latin *nil per os*)
NRTI	Nucleoside reverse transcriptase inhibitor
NS	Normal saline
NSVD	Normal spontaneous vaginal delivery
NTE	Not to exceed
O & P	Ova & parasites
ODD	Oppositional defiant disorder
OLT	Orthotopic liver transplant
OME	Otitis media with effusion
OTC	Over-the-counter
PC	Pressure control
PCA	Patient-controlled analgesia
PCDAI	Pediatric Crohn Disease Activity Index
pCO_2	Partial pressure of carbon dioxide
PCP	Primary care provider
PCV	Pneumococcal conjugate vaccine

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APPENDIX 2 (cont'd)

Abbreviation	Medical Terminology
PDA	Patent ductus arteriosus
PE	Physical examination; pulmonary embolism
PEEP	Positive end-expiratory pressure
PEF	Peak expiratory flow
PERRLA	Pupils equally round and reactive to light and accommodation
PICC	Peripherally inserted central catheter
PICU	Pediatric intensive care unit
PIV	Peripheral IV
PMN	Polymorphonuclear neutrophil
PNA	Postnatal age
po	By mouth, orally (from Latin *per os*)
pO_2	Partial pressure of oxygen
POTS	Postural orthostatic tachycardia syndrome
PPD	Packs per day (of cigarettes smoked); purified protein derivative of tuberculin
PR	Per rectum
pRBCs	Packed red blood cells
pRIFLE	Pediatric RIFLE criteria: risk, injury, failure, loss, end stage
prn	As needed (from Latin *pro re nata*)
PS	Pressure support
PT	Prothrombin time
PTA	Prior to admission
q	Every
QTc	QT interval, corrected for heart rate
RA	Room air
RBC	Red blood cell
RDS	Respiratory distress syndrome
RDW	Red cell distribution width
rhGH	Recombinant human growth hormone
RLL	Right lower lobe
RNA	Ribonucleic acid
ROM	Range of motion
RR	Respiratory rate
RRR	Regular rate and rhythm
RSV	Respiratory syncytial virus
RUL	Right upper lobe (of lung)
SBFT	Small bowel follow-through
SC	Subcutaneous
SCD	Sickle cell disease
SCr	Serum creatinine
SDS	Standard deviation score
sec	Second
SGA	Small for gestation age
SGOT	Serum glutamic-oxaloacetic transaminase
SGPT	Serum glutamic-pyruvic transaminase

Abbreviation	Medical Terminology
SIMV-PC	Synchronized intermittent mandatory/mechanical ventilation—pressure controlled
SNRI	Serotonin norepinephrine reuptake inhibitor
SOB	Shortness of breath
SpO_2	Oxygen saturation via pulse oximetry
SS	Sliding scale
SSI	Supplemental Security Income
SSRI	Selective serotonin reuptake inhibitor
SVT	Supraventricular tachycardia
T2DM	Type 2 diabetes mellitus
T_4	Thyroxine
Tdap	Diphtheria, tetanus, and pertussis
TG	Triglyceride
TM	Tympanic membrane
TMP-SFX	Trimethoprim–sulfamethoxazole
TNF	Tumor necrosis factor
tPA	Tissue plasminogen activator
TPN	Total parenteral nutrition
TSH	Thyroid-stimulating hormone
UC	Ulcerative colitis
UFH	Unfractionated heparin
UGI	Upper gastrointestinal
UOP	Urinary output
UTD	Up to date
UTI	Urinary tract infection
VAS	Visual analog scale
VLBW	Very low birth weight
VPA	Valproic acid
WBC	White blood cell
WDWN	Well developed, well nourished
WNL	Within normal limits
WPW	Wolff-Parkinson-White
wt	Weight
yr	Year

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APPENDIX 3: "DO NOT USE" ABBREVIATIONS LIST

Do Not Use	Potential Error	Use Instead
U, u	Mistaken for zero, "4" or "cc"	Unit
QD, QOD	Mistaken for each other or "QID"	Daily or every other day
1.0 or .1	Decimal point is missed	1 mg or 0.1 mg[a]
MS	Can mean morphine or magnesium sulfate	Morphine sulfate or magnesium sulfate
MSO_4 & $MgSO_4$	Mistaken for each other	

[a]A trailing zero (X.0) can be used in cases of laboratory results or other situations in which it is important to show the level of precision of the test; however, never when ordering or documenting medication doses.

Source: http://www.jointcommission.org/assets/1/18/Do_Not_Use_List.pdf. Updated June 2013.

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APPENDIX 4: SOAPE Outline—Multiple Days or Visits

DEMOGRAPHIC INFORMATION

Patient name	**Caregiver name/relationship**
Patient ID#	**DOB**
Patient MR#	**Gender**
Room#	**Height/weight**
Admission date/initial visit	**Insurance/ethnicity**

Allergies *(include reaction)*: _____

_____

SUBJECTIVE

CC _____ FHx _____

HPI _____ SHx _____

PMH _____ ROS _____

IMMUNIZATIONS: _____

_____

OBJECTIVE

Medications

	Problem	Current Pharmacotherapy (dose, formulation, route, frequency)	Assessment
Healthcare Problem List			

Physical Exam (pertinent findings from H&P)

Vitals/Labs/Dx Tests

Date												
Weight												
Vitals												
Temp												
BP												
HR												
RR												

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APPENDIX 4 (cont'd)

Date												
Chemistry												
Na												
K												
Cl												
CO_2												
BUN												
SCr												
CrCl												
Drug levels												
CBC/Coags												
WBC												
Plt												
Hgb												
HCT												
aPTT												
PT/INR												
Cultures												
Other												
Dx tests												
Eating? Tube Feeds?												
Formula amount												

Consultations

Date	Specialty	Physician

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APPENDIX 4 (cont'd)

Pertinent HCP Assessments and Plans

Date	Specialty	Assessment	Plan

Problem 1: _____

ASSESSMENT

- Proposed etiology
- Current therapy
- Evaluation of current therapy or need for additional therapy

PLAN/RECOMMENDATION

- Goals of therapy
- Recommended therapy *(including OTC and immunizations)*
- Recommended nonpharmacologic therapy
- Therapeutic endpoints/monitoring/follow-up plan
- Drugs to avoid *(and justification)*

EDUCATION

- Medications *(focus on medication administration, adherence, anticipated therapeutic and adverse reactions)*
- Nonpharmacologic therapy education

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APPENDIX 4 (cont'd)

Problem 2: _____

ASSESSMENT

- Proposed etiology
- Current therapy
- Evaluation of current therapy or need for additional therapy

PLAN/RECOMMENDATION

- Goals of therapy
- Recommended therapy *(including OTC and immunizations)*
- Recommended nonpharmacologic therapy
- Therapeutic endpoints/monitoring/follow-up plan
- Drugs to avoid *(and justification)*

EDUCATION

- Medications *(focus on medication administration, adherence, anticipated therapeutic and adverse reactions)*
- Nonpharmacologic therapy education

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APPENDIX 5: SOAPE Outline—Single Day or Visit

DEMOGRAPHIC INFORMATION

Initials: _____

DOB: _____

Ht/Wt _____

Date and time of initial visit: _____

Allergies *(include reaction)*: _____

_____

SUBJECTIVE

CC _____ FHx _____

HPI _____ SHx _____

PMH _____ ROS _____

IMMUNIZATIONS: _____

_____

OBJECTIVE

Medications

	Problem	Current Pharmacotherapy (dose, formulation, route, frequency)	Assessment
Healthcare Problem List			

Physical Exam (pertinent findings from H&P)

Vitals/Labs/Dx Tests

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APPENDIX 5 (cont'd)

Pertinent HCP Assessments and Plans

Date	Specialty	Assessment	Plan

Problem 1: _____

ASSESSMENT

- Proposed etiology
- Current therapy
- Evaluation of current therapy or need for additional therapy

PLAN/RECOMMENDATION

- Goals of therapy
- Recommended therapy *(including OTC and immunizations)*
- Recommended nonpharmacologic therapy
- Therapeutic endpoints/monitoring/follow-up plan
- Drugs to avoid *(and justification)*

EDUCATION

- Medications *(focus on medication administration, adherence, anticipated therapeutic and adverse reactions)*
- Nonpharmacologic therapy education

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Account: s8879438

INDEX

A

Abbreviations, 5–6, 8
  common, 257–259
  "do not use" list, 260
Abscess, 87–89
Absence seizures, 81–83, 205–207
  alternative agents, 206
  first-line agents, 205–206
  pharmacologic regimens, 206–207
Accountability, 1
Acute chest syndrome, 108–10, 242–46
Acute kidney injury, 52–55, 167–172
  assessment based on creatinine clearance, pRIFLE and UOP, 168
  background information, 52
  diagnostic tests, 53
  fluid and electrolytes, 170–171
  laboratory data, 54
  management goals, 168
  medication history, 53
  medications since admission, 53, 55
  minimizing nephrotoxic medications on exposure, 169–170
  monitoring parameters, 171–172
  pharmacotherapeutic plan, 168–169
  physical exam at admission, 53
  potential causes, 167
  problem list, 55
  self-assessment questions, 55
  subjective/objective evidence, 167–168
Acute otitis media, 85–86, 118, 209–212
  background information, 85
  diagnostic tests, 86
  initial observation or immediate pharmacologic treatment, 210
  medication history, 86
  medication-related problem resolution, 210
  parent education, 211
  pharmacologic therapy assessment, 118
  pharmacologic treatment plan, 210–211

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physical exam, 85–86
problem list, 86
risk factors, 209
self-assessment questions, 86
subjective/objective evidence, 209
Acute pulmonary exacerbation, 36–39, 143–47
Adolescents, 11–12
Aggressive behaviors, increase in, 69–71
Agitation/aggression, 72–74, 190–194
Alert fatigue, 5
Allergies, 4, 6
Ambulatory medical record, 3–4
American Academy of Pediatrics, 2
American Society of Health-System Pharmacists (ASHP), 5
Amoxicillin, allergic reaction to, 17–19, 117–119
Anaphylaxis, 17–19, 118–120
acute otitis media pharmacologic therapy assessment, 118
amoxicillin reaction medical documentation, 119–120
background information, 17
discharge plan, 117–118
laboratory data, 18–19
medication history, 18
medications at admission, 19
patient/caregiver education, 118–119
physical exam, 17–18
problem list, 19
self-assessment questions, 19
subjective/objective evidence, 117
Anemia, 105–107, 238–241, 244
background information, 105–106
current, desired weight classification, 240–241
laboratory data, 106
medication history, 106
medication-related problem, solution, 240
nonpharmacologic, pharmacologic regimen, 239–240
patient/caregiver education, 241
physical exam, 105–106
problem list, 106
self-assessment questions, 106–107
subjective/objective evidence, 238
Anticoagulation, 111–113, 247–251
appropriate monitoring, 248–249
background information, 111–112
laboratory data, 112
medications at admission, 113
medications dosing with sepsis, acute kidney injury, 249–250
outpatient therapy and medication-related problem management, 250
pharmacologic regimen, 247–248
physical exam, 112
problem list, 113
self-assessment questions, 113
subjective/objective evidence, 247
Anxiety, 75–77, 195–198
Arterial thrombosis, 111–13, 247–51
Assessment, 8–9, 12
Asthma, 33–35, 139–141
acute pharmacologic therapy, 140
background information, 33–34
classification of asthma control, 140
diagnostic tests, 35
discharge pharmacologic regimen, 140–41
laboratory data, 34–35
medication history, 34
medications at admission, 35
patient/cargegiver education, 141
physical exam, 34
potential medication-related adverse reaction, 140
problem list, 35
risk factors for ICU admission, 139
self-assessment questions, 35
subjective/objective evidence, 139
Attention deficit hyperactivity disorder, 69–71, 187–189
alpha-2 agonists benefits, considerations, 188
background information, 69–70
current pharmacologic therapy, 187–188
environmental factors, 187
laboratory data, 70
medication history, 70
medication-related problem, 189
patient/caregiver counseling, 188–189
pharmacologic treatment recommendation, 188
physical exam, 70
problem list, 71
self-assessment questions, 71
subjective/objective evidence, 187
Autism spectrum disorder, 72–74, 190–194
background information, 72–73
diagnostic tests, 73
laboratory data, 73–74
medication history, 73
medication-related problem, 191
nonpharmacologic and pharmacologic therapy, 190–191
patient/caregiver education, 194
pharmacotherapeutic regimen, 193
physical exam, 73
problem list, 74
seizure disorder, 181
seizure disorder alternative plan, 191–192
self-assessment questions, 74
subjective/objective evidence, 190
treatment outcomes, goals, 192–193

B

Babico, Mary A., 23–25, 126–129
Backup plan, 9
Bacterial meningitis, 102–104, 233–237
Benavides, Sandra, 1–13, 20–22, 49–51, 57–59, 60–62, 66–68, 120–125, 163–166, 173–175, 176–182, 183–186
Birth history, 7
Bloody stools, 46–48
Blue lips, 31–32
Breathing
difficulty, 17–19
inability, 22–35
Bronchopulmonary dysplasia, 40–42, 148–152
background information, 40
caregiver education for discharge, 152
diagnostic tests, 42
laboratory data, 41
medication history, 41
medication-related problems, solution, 151
pharmacologic regimen, 150–151
physical exam, 41
prevention in premature neonates, 149–150
problem list, 42
self-assessment questions, 42
subjective/objective evidence, 148

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C

Caballero, Joshua, 69–71, 187–189
Calcium carbonate, 8–9
Carbuncles, 87–89, 213–215
Cardiovascular
  congenital heart defects, 31–32, 136–138
  hypertension, 29–30, 133–135
  patent ductus arteriosis, 27–28, 131–132
  premature disease, 57–59
Caregiver interview, 4
Cellulitis, 87–89, 213–215
Cephalosporin reaction, 17–19, 117–119
Cerebral palsy, 78–80, 199–202
  background information, 78–79
  diagnostic tests, x–rays, 80
  drooling treatment, strategies, 201
  intrathecal baclofen therapy suitability, 200
  laboratory data, 80
  low bone mineral density treatment, 201–202
  medication error identification, prevention, 202
  medication history, 79
  medications at admission, 80
  parental counseling, 200–201
  physical exam, 79
  problem list, 80
  self-assessment questions, 80
  subjective/objective findings, 199–200
Cerebrospinal fluid results, 102–104
Chan, Dominic, 85–86, 92–94, 209–212, 220–222
Chest pain, left-sided, 108–110, 242–246
Chief complaint (CC), 6
Children
  2 to 7 years, 11
  7 to 11 years, 11
Clinical interventions, 5
Close-ended question, 10, 12
Cognitive development, 10–11
Communication, 10–11
Community pharmacist medical record, 4
Community-acquired pneumonia, 92–94, 220–222
Comprehension evaluation, 12
Concrete operational cognitive development, 11
Congenital heart defects, 31–32, 136–138
  background information, 31
  diagnostic tests, 32
  laboratory data, 32
  medication history, 31
  medications at admission, 32
  patient/caregiver education, 137
  pharmacologic regimen for treatment of hypercyanotic spell, 137
  pharmacologic regimen to prevent hypercyanotic spell, 136
  physical exam, 31–32
  problem list, 32
  propranolol dosage, 138
  self-assessment questions, 32
  subjective/objective evidence, 136
Constipation, 43–45, 123, 153–156
  alternative oral agents, 155
  alternative rectal agents, 155
  background information, 43–44
  classification, possible causes, 153
  diagnostic tests, 44
  laboratory data, 44
  medication history, 44
  medication-related problems, 156
  nonpharmacologic treatment options, 154
  OTC treatment, 153–54
  patient/caregiver education plan, 156
  pharmacologic treatment options, 154–55
  physical exam, 44
  problem list, 44
  self-assessment questions, 45
  subjective/objective signs, 153
Continuity of care, 9
Crain, Julianna, 66–68, 183–186
Crohn disease, 46–48, 159–161
  background information, 46
  diagnostic tests, 48
  efficacy and safety monitoring parameters, 160
  immunizations, 160–61
  laboratory data, 47–48
  medication history, 47
  medications at admission, 48
  nonpharmacologic treatment options, 160
  pharmacologic management, 158
  pharmacologic plan for remission, 158–59
  physical exam, 47
  potential interventions for medication-related problems, 159–60
  problem list, 48
  risk factors, 157–58
  self-assessment questions, 48
  subjective/objective evidence, 157
Cystic fibrosis, 36–39, 143–147
  antimicrobial therapy, 144–145
  background information, 36–37
  diagnostic tests, 39
  laboratory data, 38
  medication history, 37
  medication-related problems/solutions, 147
  medications at admission, 39
  nonpharmacologic therapy, 143–144
  nutrition/GI drug therapy, 146
  pharmacologic regimen, 144–145
  physical exam, 37
  problem list, 39
  pulmonary drug therapy, 145–146
  self-assessment questions, 39
  sputum culture, 38
  subjective/objective evidence, 143

D

Data collection sheet, 254–256
Dehydration, 20–22, 120–124
Delayed growth, 60–62
Demographic patient data, 6
Depression, 75–77, 195–197
  background information, 75–76
  follow-up schedule, 196
  laboratory data, 76–77
  medication history, 76
  medication-related problems, solutions, 197
  nonpharmacologic therapy, patient education, 196–197
  pharmacologic therapy, 195–196
  physical exam, 76
  problem list, 77
  screen, 75
  self-assessment questions, 77
  subjective/objective evidence, 195
  treatment plan, 195–196, 197
Diabetic ketoacidosis, 66–68, 183–186

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Account: s8879438

avoiding, 185
background information, 66
home medication history, 67
immunizations, 186
laboratory data, 67–68
medication-related problems, 186
medications at admission, 68
necessary laboratory parameters, 183–184
physical exam, 67
problem list, 68
self-assessment questions, 68
subjective/objective evidence, 183
therapeutic goals, pharmacologic regimen, 184–85
Diagnostic tests (Dx tests), 7, 8
Diarrhea, 46–48
Disease state, 7
identification, 2
management, 1
Documentation, 5
Drug therapy assessment, 1

E

Ear pain with fever, 85–86, 209–212
Education, 9, 10
Electronic health records (EHRs), 5
Endocrine
diabetic ketoacidosis, 66–68, 183–186
hypothyroidism, 60–62, 176–182
type 2 diabetes mellitus, 57–59, 173–175
Epilepsy, 4
Erysipelas, 87–89, 213
Even, Nicole M., 17–19, 117–119

F

Family history (FHx), 6, 7
FARM, 6
Fatigue, 46–38, 52–55
increased, 60–62
Febrile seizures, 81–83, 205, 207
Feeding, poor, 27–28
Fever, 102
persistent, 92–94, 220–222
worsening, 52–55
Folliculitis, 87–89, 213–215
Formal operational cognitive development, 11
Furuncles, 87–89, 213–215

G

Gastrointestinal
constipation, 43–45, 153–156
Crohn disease, 46–48, 157–161
Gastrostomy tube feedings, unable to tolerate, 20–22
Girand, Heather L., 90–91, 99–101, 102–04, 216–219, 227–232, 233–237
Global assessment, 2
Glycopyrrolate, 123
Growth charts, boys, 2 to 20 years
body mass index-for-age percentiles, 65, 182
stature-for-age percentiles, 64, 181
weight-for-age percentiles, 63, 180
Growth hormone deficiency, 60–66

H

Hagemann, Tracy M., 105–107, 108–110, 239–241, 242–246
Head lice, 90–91, 216–19
Heart failure, 131
Hematology/oncology
anemia, 105–107, 239–241
anticoagulation, 111–113, 247–151
sickle cell disease, 108–110, 242–246
History of present illness (HPI), 6, 7
Human immunodeficiency virus infection, 4, 99–101, 227–232
appropriate antiretroviral therapy, 229–230
background information, 99–100
diagnostic tests, 101
laboratory data, 100
medication history, 100
medication-related problem/management, 232
medications at admission, 101
monitoring plan, 230–231
physical exam, 100
pneumonia treatment plan, 227–228
problem list, 101
self-assessment questions, 101
subjective/objective evidence, 227
Hypercyanotic spells, 31–32, 136–138
Hypertension, 29–30, 133–135
background information, 29
considerations before pharmacotherapy, 133–34
diagnostic tests, 30
laboratory data, 30
medications at admission, 30
nonpharmacologic regimen, 133
obesity diagnosis criteria, 133
pharmacologic regimen, 134
physical exam, 29–30
problem list, 30
self-assessment questions, 30
subjective/objective evidence, 133
treatment goals and outcomes, 135
weight-based dosing issues, 135
Hypothyroidism, 60–62, 176–182
adrenal crisis, 178–179
background information, 60–61
boys, 2 to 20 years, growth charts, 63–65, 180–182
diagnostic tests, 61
growth hormone deficiency assessment, monitoring, 177
laboratory data, 61
medication-related adverse reaction for GH replacement therapy, 179
patient/caregiver education, 178
pharmacologic regimen, 176–177
physical exam, 61
problem list, 61
self-assessment questions, 62
subjective/objective evidence, 176
weight, height, BMI percentiles, 176

I

Immunization status, 2
Immunizations, 6, 7
Impetigo, 87–89
Indwelling medical devices, 7
Infectious diseases
acute otitis media, 85–86, 209–212
human immunodeficiency virus infection, 99–101, 227–232
Kawasaki disease, 95–98, 223–226
meningitis, 102–104, 233–237

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Account: s8879438

parasitic infections, 90–91, 216–219
pneumonia, 92–94, 220–222
skin and skin structure infections, 87–89, 213–215
Influenza vaccine, 2
Insomnia, 69–71
Intravenous fluids rehydration, 20–22
Iron-deficiency anemia, 105–7, 239–241
Irritability, 102

K

Kawasaki disease, 95–98, 223–226
additional diagnostic test, 224
background information, 95–96
diagnostic tests, 97
laboratory data, 97
medication history, 96
medication-related problems, resolution parent education, 225
medications at admission, 97
pharmacologic treatment, therapeutic goals, monitoring, 224–225
physical exam, 96
problem list, 97
risk factors supporting diagnosis, 224
safe medication use and future vaccinations parent education, 225–226
self-assessment questions, 97–98
subjective/objective evidence, 223
Kidney follow-up, 49–51

L

Laboratory values, 7, 8
Lamberjack, Kristen, 75–77, 195–98
Lashkar, Manar O., 33–35, 139–42
Le, Jennifer, 85–86, 87–89, 92–94, 95–98, 209–212, 213–215, 220–222, 223–226
Leg swelling, redness, 87–89, 213–15
Lethargy, 102

M

Madzhidova, Shirin, 60–62, 176–182
Malignancies, 4
Medical information, gathering, 2
Medical records
pharmacy documentation, 5
reviewing, 3–4
Medical terminology/abbreviations, 257–259
"do not use" list, 260
Medical-related problems, examples of, 3
Medication
efficacy, 2
history, 4, 6
recommendations, 6
reconciliation, 4–5
regimens, documenting, 4
Medication Administration Record (MAR), 3
Medication-related problems (MRPs) 1, 2, 5, 9
Meningitis, 102–104, 233–237
background information, 102
dexamethasone assessment, 235
diagnostic tests, 104
laboratory data, 103
medication history, 103
medication-related problem, management, 236
medications at admission, 104
parent education on prognosis, long-term outcomes, 235–36
physical exam, 103
problem list, 104
self-assessment questions, 104
subjective/objective evidence, 233
treatment plan, 234–35
Methicillin-resistant *S. aureus*, 38
Microcytic anemia, 105–7, 239–41
Moffett, Brady S., 27–28, 29–30, 31–32, 111–113, 131–132, 133–135, 136–138, 239–241, 247–251
Motivational interviewing, 10, 11
Multiagent overdose, 23–25, 126–29

N

Nahata, Milap C., 43–45, 46–48, 52–55, 153–156, 157–161, 167–172
Necrotizing, fascitis, 87–89, 213
Neonatal Infant Pain Scale, 8
Nephrotic syndrome, 49–51, 163–166
background information, 49–50
corticosteroid adverse reactions/management, 165
counseling points, 166
laboratory data, 50–51
medication history, 50
medication-related problems identification/resolution, 165
physical exam, 50
problem list, 51
renal biopsy, 51
self-assessment questions, 51
subjective/objective signs/symptoms, 163
treatment goals/outcomes, 163–64
treatment strategies, changes, 164–65
Nervousness, 75–77, 195–198
Neurology
attention deficit hyperactivity disorder, 69–71, 187–189
autism spectrum disorder, 72–74, 190–194
cerebral palsy, 78–80, 199–202
depression, 75–77, 195–198
seizure disorders, 81–83, 203–208
Nutrition, 20–21, 120–125
background information, 20–21
dehydration, 20–21, 120–125
dehydration severity determination, treatment plan, 120–121
estimated daily electrolyte requirements, 122
laboratory data, 21
medication history, 21
medication-related problem, solution, 123
physical exam, 21
PN formula development, 121–123
PN monitoring plan, 123
PN potential complications, 123
problem list, 21
self-assessment questions, 21
subjective/objective findings, 120
TPN orders, 124

O

Obesity, 57–59, 173–175
diagnosis criteria, 133
Objective data, 7–8
Oncology. *See* Hematology/oncology
Open-ended question, 10, 12
Oppositional defiant disorder, 69–71

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P

P. aeruginosa, 38
Pain
  crisis, 108–110, 242–246
  documentation, 8
Parasitic infections, 90–91, 216–219
  background information, 90
  hydrocortisone assessment for pinworm infestation, 217
  medication after office visit, 91
  medication history, 91
  medication-related problem/management, 219
  parent education, 218–219
  pharmacologic treatment plan, 217–218
  physical exam, 91
  problem list, 91
  self-assessment questions, 91
  subjective/objective evidence, 216–217
Parenteral nutrition plan, 20–22, 121–124. *See also* Nutrition
  formula development, 121–123
  monitoring, 123
  potential complications, 123
  TPN orders, 124
Past medical history (PMH), 6, 7
Patent ductus arteriosis, 27–28, 131–132
  background information, 27
  caregiver information, 132
  diagnostic tests, 28
  laboratory data, 28
  medications at admission, 28
  oral sucrose for pain management, 132
  pharmacologic regimen, 131–132
  pharmacologic therapy assessment, 131
  physical exam, 27–28
  problem list, 28
  self-assessment questions, 28
  subjective/objective evidence, 131
Patient care management, 1–13
  caregivers and, 1
  medication efficacy, safety, 2
Patient care plan
  development, 2
  documentation, 2, 5
Patient interview, 4
Patient-specific data, 1
Patient-specific treatment goals, outcomes, 2
Pediatric intensive care unit (PICU) med record, 3
Phan, Hanna, 17–19, 23–25, 33–35, 36–39, 40–42, 117–119, 126–129, 139–142, 143–147, 148–152
Physical examination (PE), 7, 8
Piaget, Jean, 11
Pinworm infestation, 90–91, 216–219
Plan, 9
Plan documentation, 9
Pneumonia, 92–94, 220–222
  background information, 92
  diagnostic tests, 94
  laboratory data, 93
  likely pathogens, associated complication, 220–221
  medication history, 93
  medication-related problem/resolution, 222
  pharmacologic treatment regimen, 221–22
  physical exam, 93
  problem list, 94
  self-assessment questions, 94
  subjective/objective evidence, 220
Preoperational cognitive development, 11
Preventive strategies, 1
Primary plan, 9
PRN medications, 4
Prusa, Christine, 75–77, 195–98
Psychiatry
  attention deficit hyperactivity disorder, 69–71, 187–189
  autism spectrum disorder, 72–74, 190–194
  cerebral palsy, 78–80, 199–202
  depression, 75–77, 195–198
  seizure disorders, 81–83, 203–208
Pulmonary
  asthma, 33–35, 139–142
  bronchopulmonary dysplasia, 40–42, 148–152
  cystic fibrosis, 36–39, 143–147
  overcirculation, 131
  respiratory distress syndrome, 40–42, 148–152

R

"READS," 10, 11
Renal
  acute kidney injury, 52–55, 167–172
  nephrotic syndrome, 49–51, 163–166
Respiratory distress syndrome, 40–42, 148–152. *See also* Bronchopulmonary dysplasia
  background information, 40
  diagnostic tests, 42
  laboratory data, 41
  medication history, 41
  neonatal risk factors, presentation, treatment, 148–149
  physical exam, 41
  problem list, 42
  self-assessment questions, 42
  subjective/objective evidence, 148
Review of systems (ROS), 6, 7
Rey, Jose A., 72–74, 81–83, 190–194, 203–208
Riskin, Jaime W., 1–13

S

Seizure disorders (tonic-clonic), 72–74, 190–194
  background information, 81
  diagnostic tests, 83
  hepatic impairment and pharmacologic regimens, 206–207
  laboratory data, 82–83
  medication history, 82
  medication-related problem management, 208
  nonpharmacologic, pharmacologic regimen, 204
  patient/caregiver information, 207–208
  pharmacologic treatment of absence seizures, 205–206
  pharmacologic treatment of febrile seizures, 205
  pharmacotherapy, desired outcomes, 207
  physical exam, 82
  problem list, 83
  self-assessment questions, 83
  subjective/objective evidence, 203–204
Self-injurious behavior, 72–74, 190–194
Sensory hypersensitivity, 72–74, 190–194

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Account: s8879438

Sickle cell disease, 108–110, 242–246
background information, 108
diagnostic tests, 110
hydroxyurea dosage, monitoring, 245
laboratory data, 109
medication history, 109
medication-related problem/solution, 245–246
medications at admission, 110
microbiology, 109
patient/caregiver education, 244–245
pharmacologic regimen, 242–244
pharmacologic therapy assessment, 244
physical exam, 108–109
problem list, 110
self-assessment questions, 110
subjective/objective evidence, 242–246
Skin and skin structure infections, 87–89, 213–215
antibiotic treatment regimen, 214
background information, 87
cellulitis diagnosis, 213
cellulitis pathogens, 214
diagnostic tests, 89
laboratory data, 88
medication history, 88
medication-related problem, 214
parent education, 214
physical exam, 88
problem list, 89
self-assessment questions, 89
subjective/objective evidence, 213
SOAPE
assessment, 8–9
education, 9, 10
format, 6
plan, 9
SOAPE notes, 6–7
objective data, 7–8
subjective data, 6–7
SOAPE outline
multiple days or visits, 261–64
single day or visit, 265–66
SOB, 52–55
Social history (SHx), 6, 7
Spasticity, 78–80, 199–202
Stultz, Jeremy S., 43–45, 46–48, 52–55, 153–156, 157–161, 167–172
Subjective data, 6

T

Tetanus booster, 2
Tetralogy of Fallot, 31–32, 136
Therapeutic set points, 9
Thomas, Jennifer E., 69–71, 187–189
Tires easily, 31–32
TITRS, 6
Toxicology, 23–25, 126–129
background information, 23
diagnostic tests, 25
laboratory data, 24–25
lorazepam assessment, 128
medication history, 24
medications at admission, 25
monitoring parameters, 128
multiagent overdose, 126–127
patient education plan, 129
pharmacologic regimen, 127–128
physical exam, 24
problem list, 25
self-assessment questions, 25
stress management, 129
subjective/objective evidence, 126
Transition of care, 4–5
Type 1 diabetes mellitus, 66–68, 183–186
Type 2 diabetes mellitus (T2DM), 57–59, 173–175
background information, 57–58
dyslipidemia, prehypertension, 173–174
laboratory data (fasting), 58
medication history, 58
medication-related problem/solution, 175
obesity treatment, 174–175
pharmacologic regimen, 174
physical exam, 58
problem list, 59
self-assessment questions, 59
subjective/objective evidence, 173

U

U.S. Pharmacopeial Convention (USP), 10, 12
Unresponsive, polysubstance overdose, 24

V

Valdes, Jose, 72–74, 81–83, 190–194, 203–208
Visual analog scale, 8
Vital signs (VS), 7, 8
Vomiting, intractable, 66–68, 102

W

Weight gain, poor, 27–28
Wong-Baker FACES Pain Rating Scale, 8
Worthington, Mary, 78–80, 199–202

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